A Dissertation on

PREVALENCE OF HYPERLIPIDEMIA AMONG HEALTHY FEMALES AND THEIR RELATIONSHIP WITH WAIST CIRCUMFERENCE AND BODY MASS INDEX

Dissertation Submitted to

THE TAMILNADU Dr.M.G.R. MEDICAL UNIVERSITY

CHENNAI - 600 032

with partial fulfillment of the regulations

for the award of the degree of

M.D. GENERAL MEDICINE

BRANCH-I

COIMBATORE MEDICAL COLLEGE
COIMBATORE
APRIL 2016

CERTIFICATE

This is to certify that the dissertation entitled "**PREVALENCE OF HYPERLIPIDEMIA AMONG HEALTHY FEMALES AND THEIR RELATIONSHIP WITH WAIST CIRCUMFERENCE AND BODY MASS INDEX."** is a bonafide research work done by Dr. NIRMALA. P, Post Graduate student in General Medicine, under my direct guidance and supervision. This is being submitted to the The Tamil Nadu Dr. M.G.R. Medical University, Chennai, in partial fulfillment of the regulations for the award of M.D. Degree in General Medicine examination to be held in April 2016. I have great pleasure in forwarding the same to The Tamil Nadu Dr. M.G.R. Medical University, Chennai, Tamilnadu, India.

Prof. Dr.USHA.S, M.D.,
Prof. and Guide
Department of Medicine,
Coimbatore Medical College

Prof.Dr.KUMAR NATARAJAN, M.D.,
Prof. and H.O.D
Department of Medicine
Coimbatore Medical College

THE DEAN
COIMBATORE MEDICAL COLLEGE
COIMBATORE

Coimbatore Medical College

COIMBATORE, TAMILNADU, INDIA - 641 014

(Affiliated to The Tamilnadu Dr. MGR Medical University, Chennai)

ETHICS COMMITTEE

CERTIFICATE

Name of the Candidate : DR. P. NIRMALA

Course : MD - GENERAL MEDICINE

Period of Study : 2013 - 2016

College : COIMBATORE MEDICAL COLLEGE

Dissertation Topic : PREVALENCE OF HYPERLIPIDEMIA AMONG HEALTHY FEMALES AND THEIR RELATIONSHIP WITH WAIST CIRCUMFERENCE AND BODY MASS INDEX.

The Ethics Committee, Coimbatore Medical College has decided to inform that your Dissertation Proposal is accepted / ~~Not accepted~~ and you are permitted / ~~Not permitted~~ to proceed with the above Study.

DEAN
Coimbatore Medical College & Hospital, Coimbatore

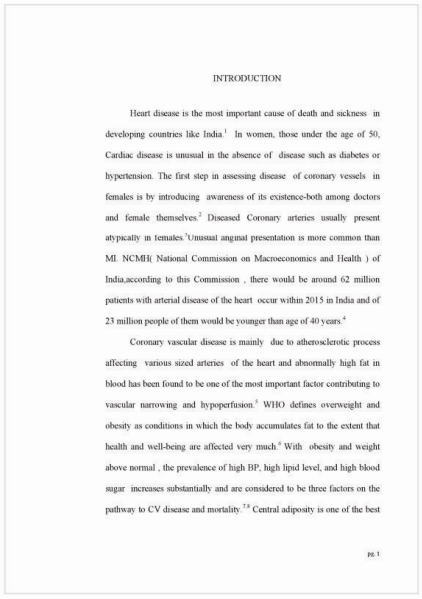
10.9.2014

Digital Receipt

This receipt acknowledges that Turnitin received your paper. Below you will find the receipt information regarding your submission.

The first page of your submissions is displayed below.

Submission author:	201311312.md General Medicine : D..
Assignment title:	TNMGRMU EXAMINATIONS
Submission title:	PREVALENCE OF HYPERLIPIDEMIA
File name:	full_project.docx
File size:	825.73K
Page count:	82
Word count:	7,679
Character count:	45,026
Submission date:	13-Sep-2015 08:33PM
Submission ID:	567628487



INTRODUCTION

Heart disease is the most important cause of death and sickness in developing countries like India.[1] In women, those under the age of 50, Cardiac disease is unusual in the absence of disease such as diabetes or hypertension. The first step in assessing disease of coronary vessels in females is by introducing awareness of its existence-both among doctors and female themselves.[2] Diseased Coronary arteries usually present atypically in females.[3]Unusual angunal presentation is more common than MI. NCMH(National Commission on Macroeconomics and Health) of India,according to this Commission , there would be around 62 million patients with arterial disease of the heart occur within 2015 in India and of 23 million people of them would be younger than age of 40 years.[4]

Coronary vascular disease is mainly due to atherosclerotic process affecting various sized arteries of the heart and abnormally high fat in blood has been found to be one of the most important factor contributing to vascular narrowing and hypoperfusion.[5] WHO defines overweight and obesity as conditions in which the body accumulates fat to the extent that health and well-being are affected very much.[6] With obesity and weight above normal , the prevalence of high BP, high lipid level, and high blood sugar increases substantially and are considered to be three factors on the pathway to CV disease and mortality.[7,8] Central adiposity is one of the best

PG 1

A Dissertation on

PREVALENCE OF HYPERLIPIDEMIA AMONG HEALTHY FEMALES AND THEIR RELATIONSHIP WITH WAIST CIRCUMFERENCE AND BODY MASS INDEX

Dissertation Submitted to

THE TAMILNADU Dr.M.G.R. MEDICAL UNIVERSITY
CHENNAI - 600 032

with partial fulfillment of the regulations
for the award of the degree of

M.D. GENERAL MEDICINE

BRANCH-I

COIMBATORE MEDICAL COLLEGE
COIMBATORE
APRIL 2016

THE TAMILNADU DR.M.G.R MEDICAL UNIVERSITY

CHENNAI

DECLARATION BY THE CANDIDATE

I, **Dr. NIRMALA.P** hereby declare that this dissertation entitled **"PREVALENCE OF HYPERLIPIDEMIA AMONG HEALTHY FEMALES AND THEIR RELATIONSHIP WITH WAIST CIRCUMFERENCE AND BODY MASS INDEX"**. is a bonafide and genuine research work carried out by me under the guidance of **Dr.USHA.S, M.D. Professor**, Department of Medicine, Coimbatore Medical College, Coimbatore, in partial fulfillment of the regulations for the award of M.D. Degree in General Medicine to be held in Apr 2016.

This dissertation has not been submitted by me on any previous occasion to any university for the award of any degree.

Date:----------
Place:Coimbatore

Dr.Nirmala.P

Post Graduate student
Department of Medicine
Coimbatore Medical College
Coimbatore

ACKNOWLEDGEMENT

I sincerely thank **Dr. A. EDWIN JOE M.D., B.L.,** Dean of Coimbatore Medical College for allowing me to utilize the hospital facilities for doing this work.

I take this opportunity to express my sincere gratitude and indebtedness to Prof. **Dr. USHA.S. M.D.,** Professor of Medicine, Department of Medicine, my unit chief for her able guidance, without whose help this study would not been possible.

I sincerely thank Prof. **Dr. KUMAR NATARAJAN, M.D.,** Head of the Department of Medicine for his suggestions throughout this study. I sincerely thank Prof. **Dr. RAVEENDRAN.M. M.D.,** for his guidance and support**.** I sincerely thank **Dr. T. GEETHA M.D., Dr. K. SIVAKUMAR M.D., Dr. P.S. RANI M.D., Dr. A.AKILA M.D.,** Assistant Professors of my unit for their thoughtful guidance.

I would like to express my gratitude to Prof. **Dr. MANIMEGALAI.N M.D.,** Department of Biochemistry for her help and guidance.

I thank my post graduate colleagues for their help and suggestions and acknowledge the co-operation of all the Staff and Technicians.

I am indebted to all the **PATIENTS** and their family members for their sincere co-operation without which this endeavour would not have been a success. I am extremely thankful to my family members for their continuous support. Above all I thank God Almighty for His immense blessings.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

ABBREVIATIONS

WHO	-	World Health Organisation
CVD	-	Cardio Vascular Disease
NCMH	-	National Commission on Macro Economics and Health
NHANES	-	National Health And Nutrition Survey
BMI	-	Body Mass Index
WC	-	Waist Circumference
WHR	-	Waist Hip Ratio
PPAR	-	Peroxisome Proliferator – Activated Receptor
FA	-	Fatty Acid
CETP	-	Cholesterol Ester Transfer Protein
PAI -1	-	Plasminogen Activator Inhibitor -1
HMG CoA Reductase	-	3 Hydroxy – 3 Methyl – Glutaryl – CoA Reductase
IL – 6	-	Interleukin – 6
TNF – ∞	-	Tumour Necrosis Factor Alpha
LP	-	Lipoprotein
LPL	-	Lipoprotein Lipase
LCAT	-	Lecithin Cholesterol Acyl Transferase
TGL	-	Triglyceride
TC	-	Total Cholesterol
VLDL	-	Very Low Density Lipoprotein
LDL	-	Low Density Lipoprotein
IDL	-	Intermediate Density Lipoprotein
HDL	-	High Density Lipoprotein
FBS	-	Fasting Blood Sugar
FH	-	Familial Hypercholesterolemia
ADA	-	American Diabetic Association

INTRODUCTION

Heart disease is the most important cause of death and sickness in developing countries like India.[1] In women, those under the age of 50, Cardiac disease is unusual in the absence of disease such as diabetes or hypertension. The first step in assessing disease of coronary vessels in females is by introducing awareness of its existence-both among doctors and female themselves.[2] Diseased Coronary arteries usually present atypically in females.[3]Unusual anginal presentation is more common than MI. NCMH (National Commission on Macroeconomics and Health) of India, according to this Commission, there would be around 62 million patients with arterial disease of the heart occur within 2015 in India and of 23 million people of them would be younger than age of 40 years.[4]

Coronary vascular disease is mainly due to atherosclerotic process affecting various sized arteries of the heart and abnormally high fat in blood has been found to be one of the most important factor contributing to vascular narrowing and hypoperfusion.[5] WHO defines overweight and obesity as conditions in which the body accumulates fat to the extent that health and well-being are affected very much.[6] With obesity and weight above normal, the prevalence of high BP, high lipid level, and high blood sugar increases substantially and are considered to be three factors on the pathway to CV disease and mortality.[7,8]

Central adiposity is one of the best predictor of Vascular Disease of the heart independently of other major risk factors.It can be considered important risk

factor when added with Body Mass Index (BMI).[9] The relationship among them is mediated by a modification /resistance produced by insulin on the metabolism lipids.[10]Individuals who presented "centrally obese" (e.g., with a high Waist circumference and WH Ratio) will have high serum lipid levels [11] These observations has been noticed in a variety of populations all over the world specific in developed[12] and less developed countries.[13]

Apart from its interest for establishing a physiopathological causal link, this predictive association suggests the possibility of measuring one or more anthropometric indices of central adiposity as a first step in population screening for high lipid level in blood.[14]

BMI when used alone as a anthropometric measurement is inadequate to differentiate between fat and fat-free individuals. Other anthropometric measurements which measures central obesity like WC, WHR should be added with BMI to assess dyslipidemia and future risk of CVD[15]

In our cross-sectional study we studied the pattern of hyperlipidemia and its correlation with Waist Circumference and Body Mass Index(BMI) among healthy, asymptomatic female patients attending out patient department in CMCH, Coimbatore.

AIM OF THE STUDY

The study aims to identify the prevalence of dyslipidemia in asymptomatic female patients attending master health checkup in Coimbatore Medical College hospital.

OBJECTIVES

- To assess the prevalence of hyperlipidemia among healthy females
- To analyze the significance between hyperlipidemia and anthropometric indices including WC,WHR and BMI.
- To correlate lipid subfractions with anthropometric measures.
- To find out the distribution of dyslipidemia in female population according to their age,physical activity and personal habits.

REVIEW OF LITERATURE

Heart Disease (CVD) is now the most common cause of death worldwide now. In 2010, Heart disease mainly coronary disease caused 16 million deaths worldwide (30%).This includes nearly 40% of deaths in developed countries and 28% in developing countries like India[16] . The overall variation in cardiac disease rates is related to ethinic, genetic, environmental and regional variations in risk behaviours. Worldwide, high lipid levels are estimated to play a role in 56% of ischemic disease of heart and 18% of, cerebrovascular accidents, accounting to 4.4 million deaths per annum. In 2008, age standardized mean total cholesterol was 179.4 mg/dl in men and 184.2 mg/dl in women[17].

Obesity is increasing throughout the world, particularly in developing countries .Three risk factors for CAD are hypertension, dyslipidemia, and cigarette smoking[18]. All these can be preventable and able to treat. In recent times, more importance has been focused on treatment of cholesterol mainly through behavioural modifications ,traditional diet and by drugs. The various drugs,i.e statins, fibrates and glitazars (PPAR modulators). Each of them, attacking metabolism of lipids at various state through its own mechanism. The HMG-CoA reductase is the enzyme which is the rate limiting step in lipid synthetic pathway. The inhibitors in this pathway (Hydroxy, Methyl Glutaryl co-A) are often called to as “statins“,continue to be the main treatment in hyperlipidemia.

PHYSIOLOGY OF LIPID METABOLISM

Lipids are consumed in the form of neutral fats which are also known as triglycerides. Triglycerides are made up of glycerol nucleus and free fatty acids. Triglycerides form the major constituent in foods in animal origin and much less in foods of plant origin. Apart from triglycerides, usual diet also contains small quantities of cholesterol and cholesterol esters.

Dietary fats are classified into two types:

1. Saturated fatty acids
2. Unsaturated fatty acids.

Saturated fats contain triglycerides which has maximum amount of hydrogen ions without any double bonds between carbon atom. It is rich in full fat milk, meat, cheese, baked biscuit, coconut oil, palm oil and deep fried fast foods.

Unsaturated FA formed by dehydrogenation of saturated FA. It can be classified into 3 types:

1. Monounsaturated fats
2. Polyunsaturated fats
3. Trans fats.

Monounsaturated fats which contains on double bond between the carbon atoms and it is rich in nuts(cashews, almonds, hazelnuts and peanuts), peanut oil and margarine.

Polyunsaturated fats which contains more than one double bond between the carbon atoms. It is rich in fruits, vegetables, walnuts, fish, sea foods and egg.

Polyunsaturated fats are of two types: 1.Omega -3 fats 2. Omega -6 fats. Both Omega -3 fats and Omega -6 fats are beneficial to the body. However, consuming too much of Omega -6 fats results in hazards than benefits.

DIGESTION OF LIPIDS

Area	Juice	Enzyme	Substrate	End product
Mouth	Saliva	Lingual lipase	Triglycerides	Fatty acid 1, 2-diacylglycerol
Stomach	Gastric juice	Gastric lipase(weak lipase)	Triglycerides	Fatty acids Glycerol
Small intestine	Pancreatic juice	Pancreatic lipase	Triglycerides	Monoglycerides Fatty acids
		Cholesterol ester hydrolase	Cholesterol ester	Free cholesterol Fatty acids
		Phospholipase A	Phospholipids	Lysophospholipids
		Phospholipase B	Lyso Phospholipids	Phosphoryl choline Free fatty acids
		Colipase	Facilitates action of Pancreatic lipase	---
		Bile-salt activated lipase	Phospholipids	Lysophospholipids
			Cholesterol esters	Cholesterol and fatty acids
	Succus entericus	Intestinal lipase	Triglycerides	Fatty acids glycerol (weak action)

TABLE1:DIGESTION OF LIPIDS IN THE BODY

Trans fats which contain trans double bond between carbon atoms and it is rich in milk, cheese, lamb and beef.

Bile salts also involved in the digestion of lipids. The final end products are FA, cholesterol and monoglycerides. They are absorbed from the micelles enters the intestinal mucosal cells by simple diffusion and re-esterified to triglycerides and cholesterol esters. They form chylomicrons which cannot pass through membranes of the capillaries because of its larger size they enter the lymphatic lymph vesssels. The lipids are stored in the adipose tissues in the form of triglycerides. Obesity does not depend on the body weight but on the amount of body fat.

ADIPOSE TISSUE

Adipose tissue or fat is a loose connective tissue that forms the storage site of fat in the form of triacylglycerol.Adipose tissue is of two types, white and brown adipose tissue.

WHITE ADIPOSE TISSUE

It is distributed underneath the skin ,forms subcutaneous fat and also surround the internal organs.These fat cells are unilocular i.e contain one single large vacuole filled with fat.It has three functions1.storage of energy which is under the control of insulin.2.Heat insulation.3.Protection of internal organs by acting as mechanical insulator.

BROWN ADIPOSE TISSUE

It is a specialized form which has the function opposite to white adipose tissue.It is present over the areas in the back of neck and inter scapular region . It is forming 5% of total tissue during infancy ,disappears gradually and forms only 1%of total adipose tissue in aduts.These cells are multilocular.i.e,contains many small vacuoles flied with fat.The colouration is due to large number of iron rich mitochondria.It function are 1.nonshivering thermogenesis-very essential in infants.2.functions as mitochontrial uncoupling of protein and produces heat and these are called as thermogenin.

PATHOPHYSIOLOGY OF ADIPOSE TISSUE

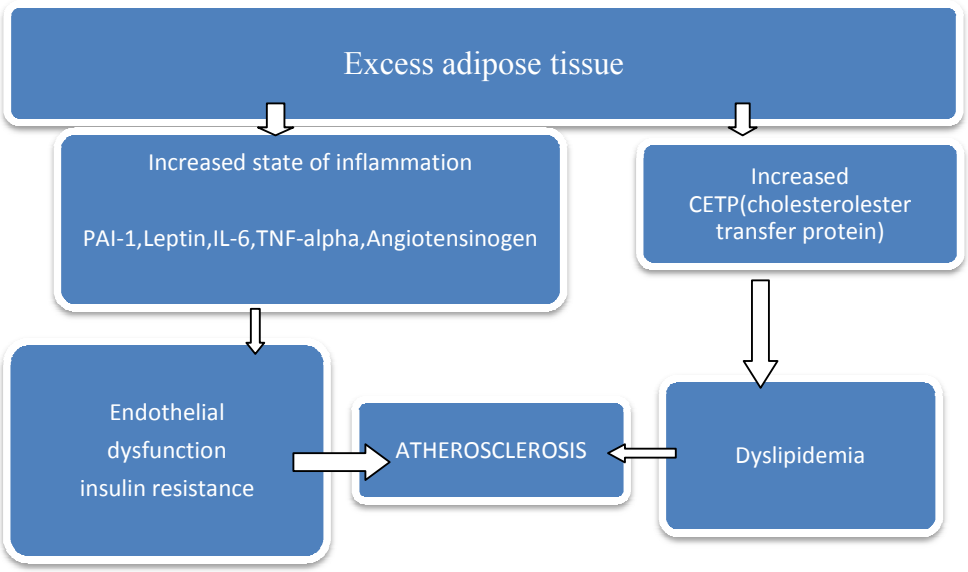


FIG 1: ROLE OF ADIPOSE TISSUE IN DYSLIPIDEMIA

True adipokines such as leptons and adiponectin, are produced exclusively by adipose tissue.

Visceral adipose tissue CETP, PAI-1,angiotensinogen, adiponectin and IL-6. Obesity can be due to chronic low grade inflammation characterized by elevated plasma levels of inflammatory markers. Except adiponectin which is low in obese persons. This state of inflammation may contributes to health problems associated with obesity such as dyslipidemia, insulin resistance and atherosclerosis.

TABLE 2: FUNCTIONS OF ADIPOKINES

Adipokines	Functions
Adiponectin	Anti-inflammatory, Insulin sensitizer
Leptin	Regulate appetite and energy expenditure
Platelet activator inhibitor-1	Contributes to the prothrombotic state, proinflammatory
TNF-alpha	Impairs the insulin signaling cascade, Inducing insulin resistance Contributes to proinflammatory state
IL-6	Stimulates CRP release from the liver Causes insulin resistance Contributes to proinflammatory state

PATHOPHYSIOLOGY OF LIPID METABOLISM

Cholesterol Homeostasis

Cholesterol is an essential cell membrane component and it is derived from steroid hormones and bile acids. Sources of cholesterol are produced by the body itself and from ingested foods, which is necessary to carry out normal functioning of life.Blood cholesterol levels approximately 40-60% from endogenous source, remaining from dietary sources.

Triglycerides are synthesized in the body mainly from diet rich in carbohydrates . In the liver ,the fatty acids can be esterified with glycerol and stored[19]. Lipids, triglycerides and cholesterol are transported in the blood by combining with lipoproteins which are spherical in its nature.These Lipoproteins (LP) depending on their composition it was categorized into five major classes[20].

Among the classes, LDL constitutes for approximately 60%-70% of total serum cholesterol. HDL constitutes 20-30% of total serum cholesterol and VLDL constitutes about 10-15%.Vascular diseases of the heart are the most prevalent and preventable cause of death and disability both in developed as well as developing countries.The higher death in CAD found among South Asians. According to NMCH, (National Commission on Macroeconomics and Health) ,In India the prevalence of CAD by 2015 would be around 62 million and of these, 23 million would be patients less than 40 yrs of age group. Dyslipidemia causes atherosclerotic deposition of large and medium sized arteries. It has been found to be one of the most contributing factor for CAD.

According to size of particle size, Chylomicrons are the largest one. Intermediate density lipoprotein (IDL), Low Density Lipoprotein (LDL) ,High Density Liporotein (HDL)are smaller in that order. The particles which are larger one have TGL rich ,the smaller density particles have cholesterol ester rich core[21].

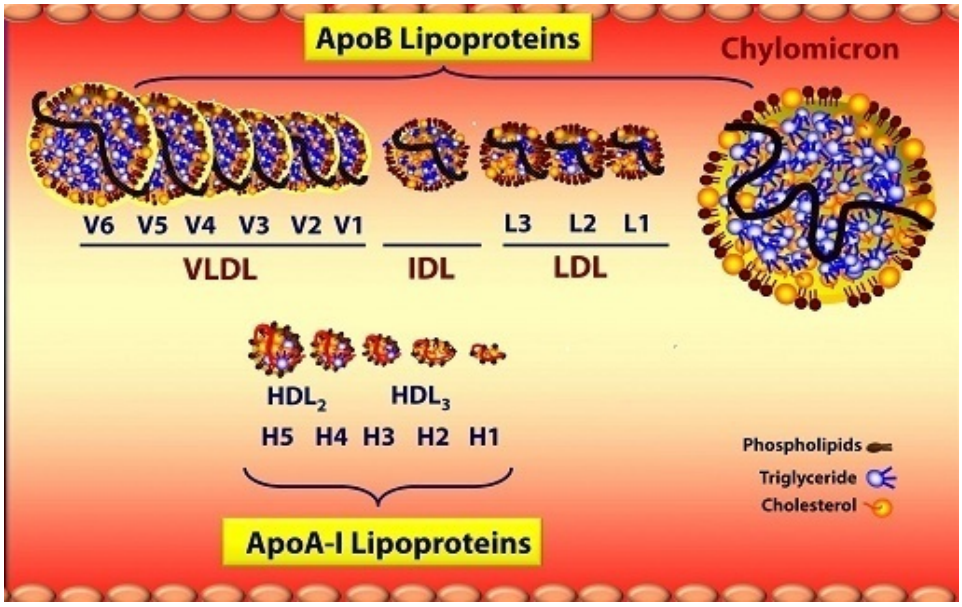


FIG 2: STRUCTURE OF LIPOPROTEINS

Category	Composition	Origin
Chylomicrons	Exogenous triglycerides	Gut
Very low density lipoproteins	Triglycerides	Liver
Intermediate density lipoproteins	Cholesterol esters and triglycerides	VLDL/HDL
Low density lipoproteins	Cholesterol	VLDL catabolism
High density lipoproteins	Cholesterol	Liver, gut

TABLE 3: CHARACTERISTICS OF THE MAJOR CLASSES OF LIPOPROTEINS.

Functions of lipoproteins;

Lipoproteins are involved in transport of free fatty acids in blood in combination with albumin.

LIPOPROTEINS	FUNCTIONS
VLDL	Transports Triglycerides from liver to adipose tissue
IDL	Transports Triglycerides ,Cholesterol and phospholipids from liver to peripheral tissue
LDL	Transports Cholesterol and phospholipids from liver to tissues and organs like Heart
HDL	Transports Cholesterol and phospholipids from tissues and organs like Heart back to Liver

TABLE 4: FUNCTIONS OF LIPOPROTEINS.

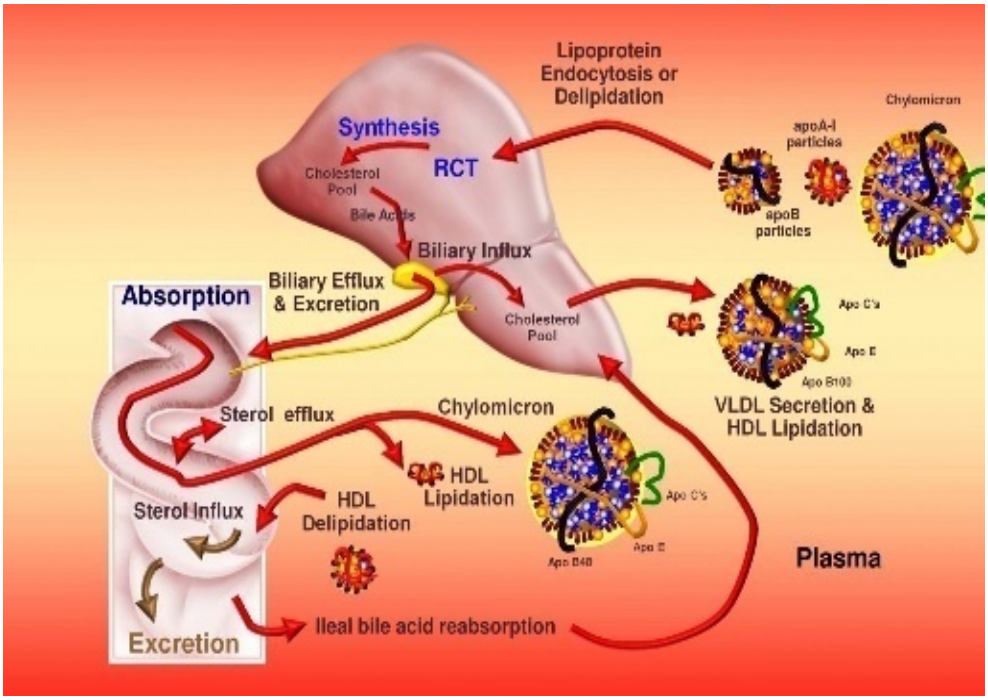
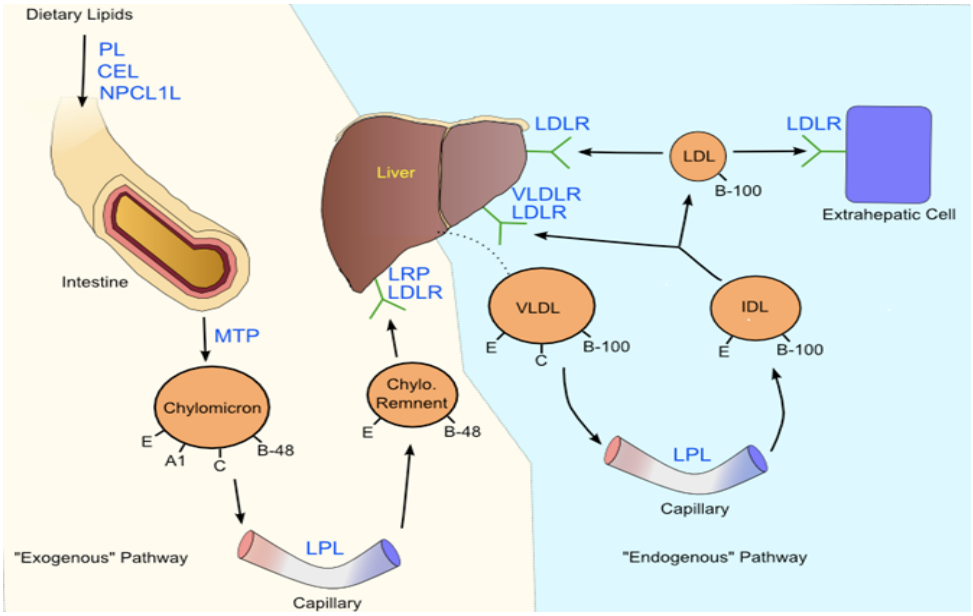


FIG 3: SOURCES OF CHOLESTEROL

1. Exogeneous cholesterol derived from systemic circulation.

2.Endogenous cholesterol synthesized within the cell[22].



In the first pathway, dietary fat is digested ,then absorbed in proximal part of small intestine.After absorbtion ,they are transformed to chylomicrons which are made up of fatty acids, cholesterol, and apolipoproteins.

In the endogenous pathway, lipoprotein are mainly involved in the synthesis, transport in blood and catabolism of chylomicron particles and remnants[23]. They are all reformulated in the intestinal endothelium where they are all packed into These chylomicrons then enter and transported via the lymphatic system.In the capillary beds,it can be metabolized by the enzyme called lipoprotein lipase (LPL).

These chylomicrons acted upon by lipoprotein lipase break into chylomicron remnants which contain less fatty acids and apolipoprotein B-48 Apo-E .These remnant proteins are cleared from blood via LDL receptor protein in the liver.[25]

In the second pathway, cells can synthesis their own cholesterol from acetyl- CoA through series of biochemical reaction.

.

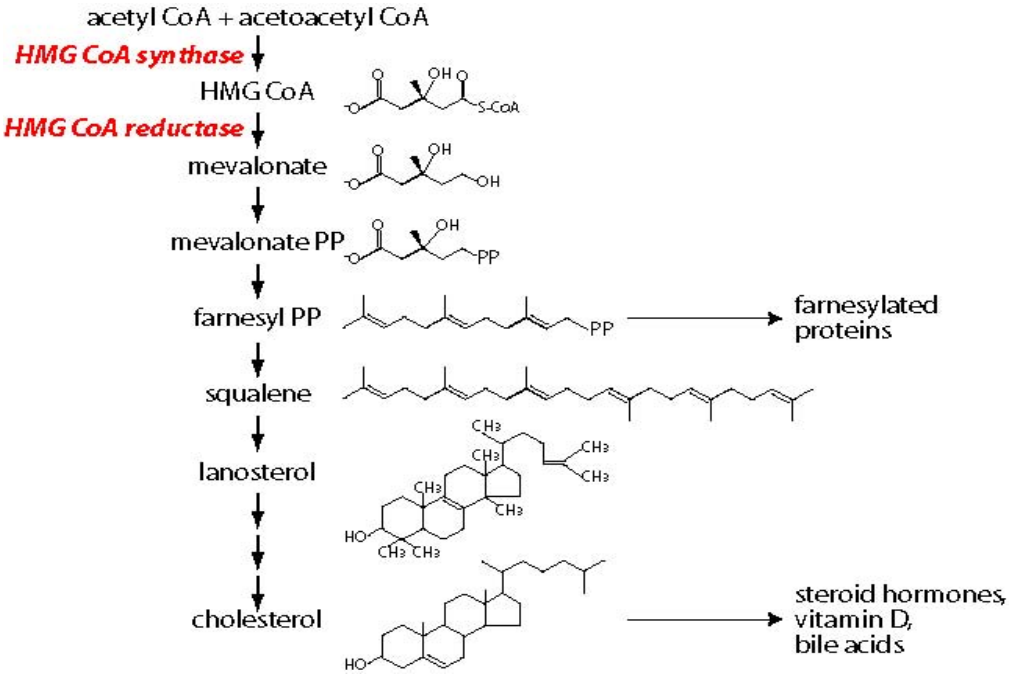


FIG 4: CHOLESTEROL SYNTHESIS PATHWAY

Acetyl-coA is converted into HMG-CoA by an enzyme called HMG-CoA synthetase.It can converted to mevalonate,then farnesyl pp ,squalene, lanosterol and finally become cholesterol through serial enzymatic reactions.The enzymes involved are HMG CoA synthase and HMG CoA reductase .

The inhibitors of HMG-CoA reductase reduce the production of cholesterol in the cell,by competitively inhibiting this enzyme[23].

VLDL, LDL, HDL. are the three major lipoprotein involved in endogenous pathway.

Cholesterol and fatty acids enter the endogenous pathway. Triglycerides produced in the presence of excess of carbohydrates are secreted into blood stream (VLDL). These contain

- apolipoprotein B-100,
- apoproteins E,
- apoproteins C-11 along with triglycerides and cholesterol. These VLDL contains five times more TGL than cholesterol.

Apoprotein B and E - link with B-E or LDL cell surface receptors. Apolipoprotein C-II - cofactor for the enzyme lipoprotein lipase (LPL).

The FFA are used for energy production primarily by cardiac and skeletal muscle and stored in fat cells.

FIG 5:FATE OF TRIGLYCERIDES

Triglyceride molecules hydrolysed by the enzyme LPL.This process of lipolysis decreases the content and the size of VLDL.It enters either of the two pathways

1.Hepatic Clearence

2.Transformation to IDL.

IDL contains almost all the cholesterol present in the VLDL.TGL content is high in IDL particles. LPL and Hepatic lipase, convert them into LDL (rich in cholesterol). Only apolipoproteins E and C removed.This IDL particles have shorter life span as they are intermediate products of VLDL and LDL. Cholesterol measurements are not significantly impacted by cholesterol and triglyceride contents. In IDL particles, cholesterol circulates less than 5%, except for rare dyslipidemias. IDL particles are cleared from circulation and are converted to LDL receptor and LDL.

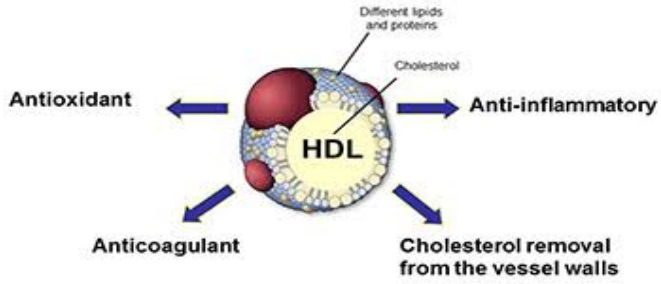
LDL penetrates into subendothelial tissue contributing to the development of atherosclerosis. LDL particle penetrates more into subendothelial tissue, when its smaller. Cholesterol gets deposited outside the cell, when there is excessive circulation of LDL cholesterol. This deposition of LDL cholesterol causes plaque formation in the endothelium and narrowing of Coronary Arteries. According to recent developments, CHD risk is highly found in two specific types of LDL particles-1.lipoprotein(a) protein.

2.atherogenic lipoprotein phenotype B, which is quite dense. This subclass has high risk of CHD and is found in approximately 30% of the population.

HDL is the third major lipoprotein that is involved in endogenous pathway. HDL particles are rich in cholesterol and are smaller in size than LDL. HDL are

involved in transport of cholesterol from intestine to liver and has antiatherogenic potential. Cholesterol that are deposited within arterial wall might be removed or prevented by HDL. Other benefits of HDL are**:**

FIG 6: FUNCTIONS OF HDL



Works as anti-oxidant and prevents LDL oxidation.

Reduces aggregation and activation of platelets by producing more prostacyclin. Facilitates fibrinolysis by stabilizing prostacyclin levels in serum. Competitively inhibits the LDL uptake by endothelium..

Aggregation and uptake of LDL by macrophages are prevented.

Production of cholesterol and foam cell is reduced.

HDL reduces with the increase in triglycerides.hence it is a marker of altered metabolism of VLDL and chylomicrons[26].

Transportation of cholesterol to the liver from the periphery involves two key enzymes and they are eliminated by HDL particles.

LCAT- lecithin cholesterol acyl transferase

IDL and VLDL are eliminated through liver.

The three lipoproteins play a role in peripheral utilization of TGL and cholesterol from the liver and vice versa for elimination.

Insufficient requirement of cholesterol by the cell will increase the production of LDL receptor and this will moves to the area on the cell surface called coated pits, where the cells identify lipoproteins which contain apo E or B present in VLDL, IDL and LDL .The binding affinity for LDL receptors are more for VLDL and IDL. Because it contains both Apo B and E. when binding occurs on the surface, internalization of the lipoprotein by cell occurs, action of liposomes takes place where it is broken down and used by the cell. The LDL receptor protein again returns comes back to the surface of the cell and the process continuously repeated.

RELATIONSHIP BETWEEN CHOLESTEROL AND CHD

The relationship between lipids and lipoproteins with coronary disease is still in controversy and research .The initiating factors of plaque formation are 1. The entry of LDL and Lp(a) into the sub-endothelial space 2. oxidatively modified free radicals produced by smooth muscle cells,

3.activated macrophages 4. Vascular endothelial cells.

These lipoproteins are oxidatively modified. It is taken up by the macrophages via scavenger receptor pathway. this process yields foam cells which are rich in lipids.

The next step is the fatty streak formation.It is formed of oxidatively modified lipoproteins and the foam cells. The muscle cells moves to subendothelial space and its proliferation occurs within the intima, ultimately leading to atherogenesis.lesions continue to grow by increased smooth muscle and collagen synthesis.At one point, foam cell undergo necrosis and extracellular lipid core forms, it triggers an autoimmune response, causing T lymphocyte infiltration into advential layer of blood vessel.this completes the process of atherogenesis,the main factor involved in CHD.

CAUSE OF ALTERED LIPID METABOLISM

By excessive secretion of VLDL

By impaired lipolysis of triglyceride rich lipoproteins

By impaired hepatic uptake apo-B containing lipoproteins

Secondary causes-liver disorders, drugs, hypothyroidism nephritic syndrome, autoimmune diseases, malnutrition, anorexia nervosa,diabetes mellitus type-2, obesity, menopause, stress, pregnancy, renal failure, acromegaly, cushing's syndrome, glycogen storage disease, acute intermittent porphyria.

SUBCLINICAL HYPOTHYROIDISM AND DYSLIPIDEMIA

Various randomized studies observe rise in total cholesterol, LDL and serum triglyceride in subclinical hypothyroid patients. This association is more strongly found in females, with increasing age and those with baseline TSH >12 mlU/ L. It also associated with increased intima and media thickness, endothelial dysfunction, systolic and diastolic dysfunction leads to cardiovascular risk. Coronary heart diseases are more with TSH >10mlU/ L[24].

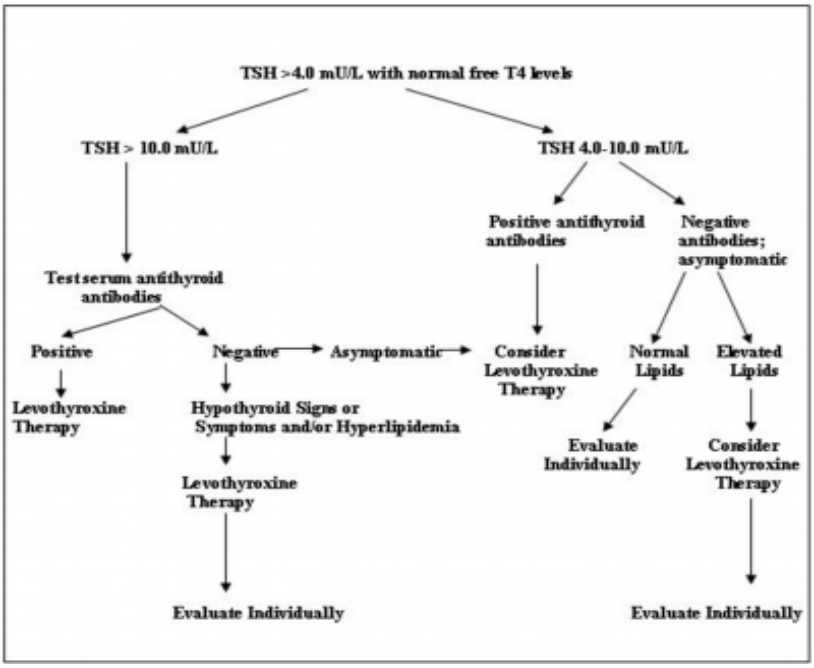
The American Thyroid Association has set guidelines for evaluation of patients with symptoms and with known thyroid problems.

Screening is suggested for

1. Women from the age of 35,
2. at 5-year intervals, for individuals with a strong family history of thyroid disease,
3. Elderly
4. Women at 4-8 weeks postpartum
5. Patients with autoimmune illnesses.

Screening of asymptomatic patients are not recommended. Early evaluation of people with even subtle signs and symptoms are encouraged.

The American College of Physicians guidelines for screening thyroid disease recommends TSH levels monitoring only in women over 50 years. Because in younger population, the prevalence of asymptomatic thyroid is low. It recommended treatment only in symptomatic and hyperlipidemic patients, with 2 to 5 yearly follow up.



CARDIOVASCULAR DISEASES AND STROKE IN WOMEN

Estrogens and androgens have major effects on the cardiovascular system and metabolism of lipids.

Estrogens-increases high-density lipoprotein lowers low-density lipoprotein (direct vasodilator effects on the vascular endothelium,

enhances insulin sensitivity antioxidant and anti-inflammatory)[26]

Androgens- have the opposite effect- decrease HDL, increases LDL. Hence the rate of CHD increases after both surgical and natural menopause. The most

common presentation of MI in women is cardiac arrest or cardiogenic shock. In men, it mostly presents as ventricular tachycardias.

Younger women has higher mortality rates than with men of similar age. This mortality gap has greatly fallen because of the advent of percutaneous transluminant coronary angioplasty. There are lower rate of restenosis and a better long term outcome.

RISK FACTORS FOR CHD IN MEN AND WOMEN:

Elevated cholesterol levels,

Low HDL cholesterol,

Obesity,

Smoking,

Lack of physical activity,

Hypertension and diabetes mellitus

IMPORTANT RISK FACTORS IN WOMEN:

Total triglyceride levels are an independent risk factor.

Low HDL cholesterol

Diabetes mellitus

Smoking-accelerates atherosclerosis and has negative effect on cardiac function, causes early menopause are more important risk factors for CHD in women than in man.

According to National Health and Nutrition Survey(NHANES), prevalence rates both coronary heart disease and stroke have increased in the 1994-2004 compared to the 1988-1994 NHANES, whereas prevalence rates have decreased or remained unchanged in men. These increases were paralleled by an increasing prevalence of abdominal obesity and other components of metabolic syndrome.

CLASSIFICATION OF DYLIPIDEMIA

Dyslipidemia may be caused by primary genetic predisposition or by secondary causes and most of the times the combination of both. There are three major forms of dyslipidemia-

Hypercholesterolemia,

Hypertriglyceridemia and

Mixed (combination of both)

In all these cases there is either increase in number or the compostion of specific lipoproteins.this is the major determinant in selecting the appropriate drug therapy.

TABLE 5:FREDRICKSON CLASSIFICATION OF HYPERLIPOPROTEINEMIAS

TABLE 356-3 Fredrickson Classification of Hyperlipoproteinemias

Phenotype	I	IIa	IIb	III	IV	V
Lipoprotein, elevated	Chylomicrons	LDL	LDL and VLDL	Chylomicron and VLDL remnants	VLDL	Chylomicrons and VLDL
Triglycerides	↑↑↑	N	↑	↑↑	↑↑	↑↑↑
Cholesterol (total)	↑	↑↑↑	↑↑	↑↑	N/↑	↑↑
LDL-cholesterol	↓	↑↑↑	↑↑	↓	↓	↓
HDL-cholesterol	↓↓↓	N/↓	↓	N	↓↓	↓↓↓
Plasma appearance	Lactescent	Clear	Clear	Turbid	Turbid	Lactescent
Xanthomas	Eruptive	Tendon, tuberous	None	Palmar, tuberoeruptive	None	Eruptive
Pancreatitis	+++	0	0	0	0	+++
Coronary atherosclerosis	0	+++	+++	+++	+/–	+/–
Peripheral atherosclerosis	0	+	+	++	+/–	+/–
Molecular defects	LPL and ApoC-II	LDL receptor, ApoB-100, PCSK9, LDLRAP, ABCG5 and ABCG8		ApoE	ApoA-V	ApoA-V and GPIHBP1
Genetic nomenclature	FCS	FH, FDB, ADH, ARH, sitosterolemia	FCHL	FDBL	FHTG	FHTG

Abbreviations: ADH, autosomal dominant hypercholesterolemia; Apo, apolipoprotein; ARH, autosomal recessive hypercholesterolemia; FCHL, familial combined hyperlipidemia; FCS, familial chylomicronemia syndrome; FDB, familial defective ApoB; FDBL, familial dysbetalipoproteinemia; FH, familial hypercholesterolemia; FHTG, familial hypertriglyceridemia; LPL, lipoprotein lipase; LDLRAP, LDL receptor associated protein; GPIHBP1, glycosylphosphatidylinositol-anchored high density lipoprotein binding protein1; N, normal

Familial Hypercholesterolemia (FH):

FH is characterized by- elevated LDL-C with normal TGL leading to premature coronary atherosclerosis ,tendon xanthomas. The elevated levels of LDL-Chloesterol in FH is due to increased synthesis and decreased catabolism of LDL. There were two mutated LDL receptor alleles Homozygote and Heterozygote. In this FH Heterozygote are less affected than FH Homozygote. According to LDL-receptor activity they can be classified into receptor negative (<2% of normal LDL receptor activity), receptor defective (2-25% of LDL receptor activity).

In Homozygote FH, total cholesterol levels are usually >500 mg/dl and even >1000 mg/dl. FH causes atherosclerosis in early life, leading to vascular diseases and death in childhood. Atherosclerosis initially develops in one of the aortic root leading to valvular stenosis and it can extends into ostia of the coronary vessels, which leads to stenosis.

In Heterozygous FH, hypercholesterolemia presents since birth, but it cannot be detected until adulthood. It produces corneal arcus, tendon xanthoma and CAD. The tendon xanthomas present over the elbows, knees, dorsum of the hands and over the Achilles tendons in~75% of patients.

FH Heterozygotes who presented with high LP (a) are associated with increased risk of vascular complications especially in heart. In untreated men it can causes MI in ~50% of persons below the age of 60years. In females it can leads to CAD ,but it can manifest only at later ages.Although the age of onset of

atherosclerotic heart disease is late in women with FH, coronary heart disease(CHD) is significantly more commen in women with FH in the Female population. No specific diagnostic is not available to detect heterozygous FH.

Hypertriglyceridemia produced by elevated levels of VLDL or chylomicron particles. It can be 1.Primary hypertriglyceridemia by the excessive production from diet

2. Secondary hypertriglyceridemia, following obesity , diabetes and other chronic medical diseases.

Hypertriglyceridemia is not an isolated one it can associated with low HDL and elevated LDL can lead to accelerated atheroscelerotic deposition in the vascular endothelium.

Mixed hyperlipidemias - most common forms of dyslipidemia presented with both triglyceride and cholesterol in high levels.

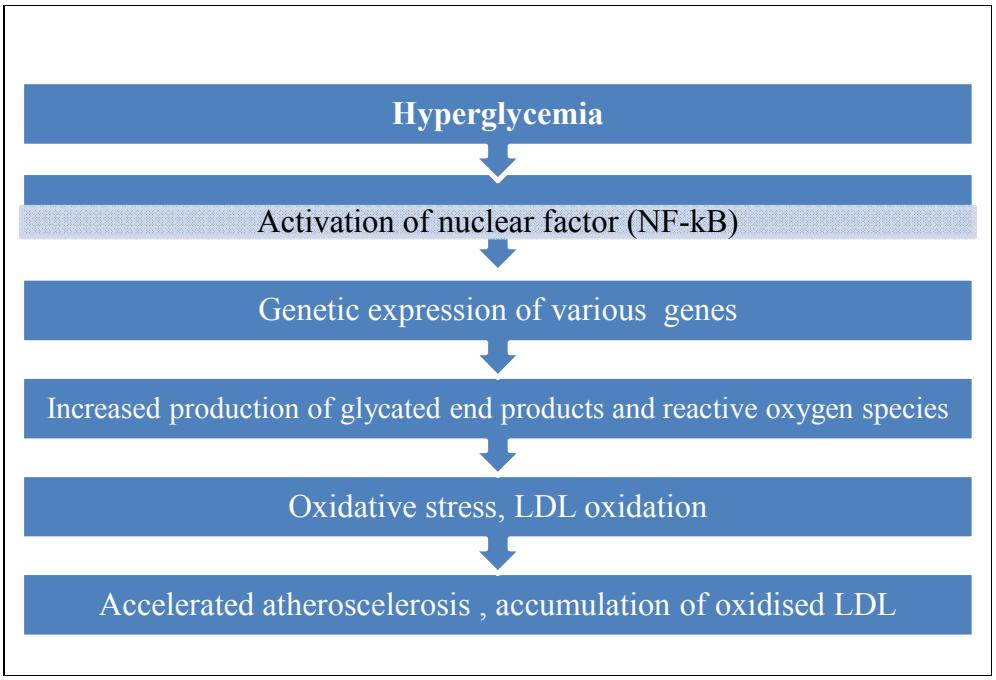
In Familial combined hyperlipidemia; It can be characterized by increased ApolipoproteinB, Overproduction of VLDL, elevated TGL and high LDL. It occurs 1 in every 100 in the population it has various presentation within the family members they can presented with hypercholesterolemia and high TGL leads to vascular disease in the heart.

Mixed hyperlipidemia also has familial lipoprotein lipase(LPL) enzyme deficiency.It has reduced delipidization of VLDL and chylomicrons and causes elevated triglyceride which leads to increased risk of pancreatitis.The aim of treatment should be to reduce triglyceride level and prevention of pancreatitis.

DYSLIPEDEMIA IN DIABETES MELLITUS.

Diabetes produces the following changes in the vascular endothelium

FIG 7: ROLE OF DIABETES MELLITUS IN ATHEROSCLEROSIS

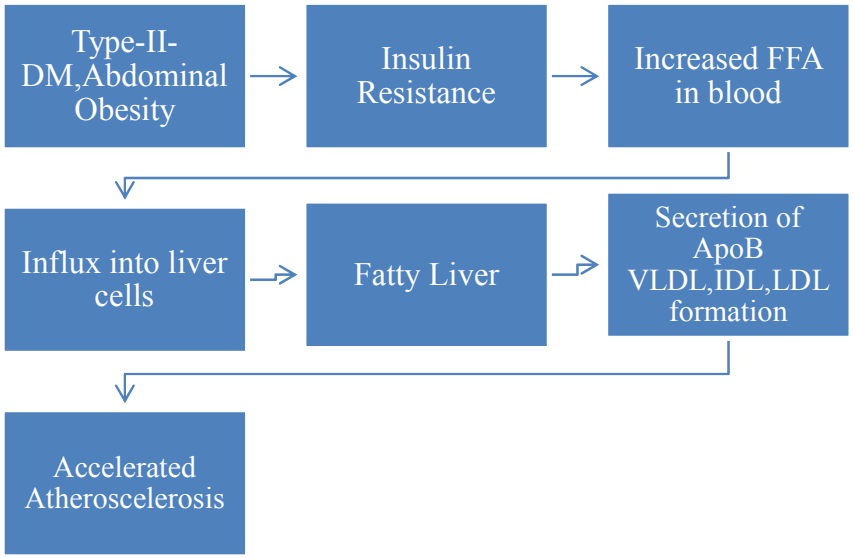


Diabetic dyslipidemia can be diagnosed by lipid profile after 10-12hours of fasting. It is characterized small dense LDL. ApoB level is more informative but its estimation is not readily available in most of the hospitals. Non HDL cholesterol estimation can be used in such situations.

Action of Insulin on lipids

Physiologically, insulin inhibits lipolysis. In insulin deficiency/resistance the following changes takes place leading to atherosclerotic disorders

FIG 8:ROLE OF INSULIN IN ATHEROSCLEROSIS



HYPERDLIPEDEMIA IN HYPERTENSION:

In hypertensive patients the following changes takes place which leads to deposition of cholesterol in blood vessels.

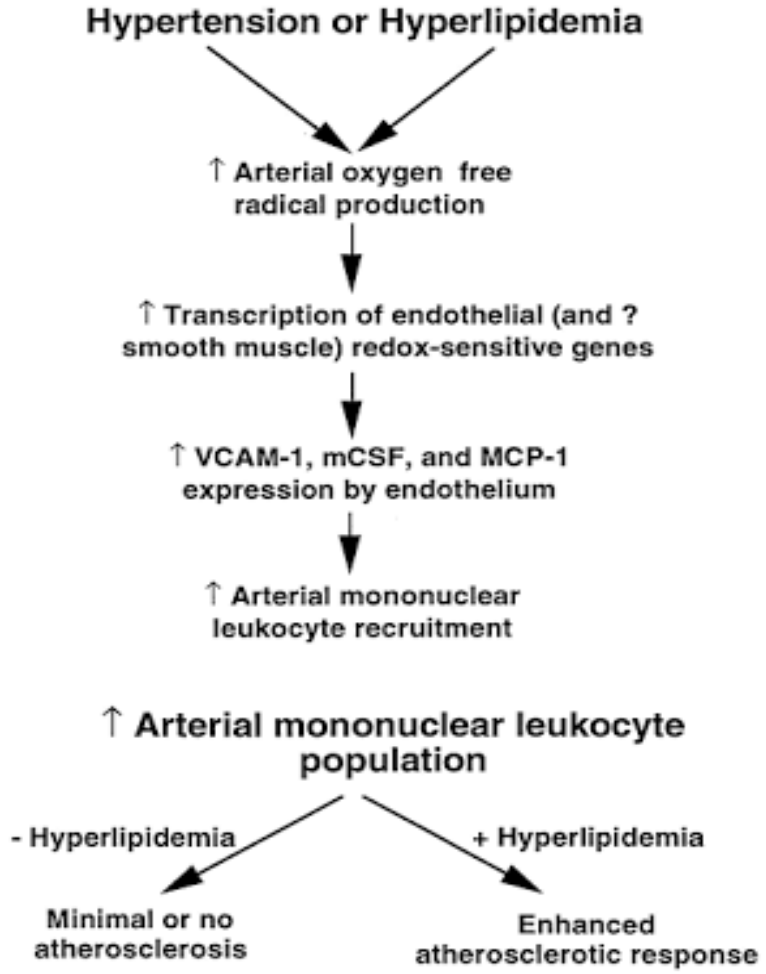


Fig : Role of Hypertension in Atherosclerosis

Women with CAD have poor prognosis because atherosclerotic plaque (made up of cellular and fibrous tissue), on erosion and rupture leads to thrombogenesis and vascular occlusion. After CABG, they had poor outcome because of relatively small coronary vessels than man, more left ventricular hypertrophy. They have atypical presentation of angina .

(microvascular), made difficulty in evaluation. Advantages of female sex can be eliminated by diabetes,obesity and distribution of fat in women can also risk factor for CAD. According to NCEP ATP III,guidelines are formed to identify and manage hyperlipidemia.

PREVENTION AND CONTROL OF OBESITY AND DYSLIPIDEMIA

According to WHO expert committee 1995 maintaining weight by prevention of weight gain of more than 5kg in all people. In those who are already overweight, 5-10% of body weight reduction is the initial goal. Prevention should begin in early childhood. It is difficult to treat obesity in adults than in children. This can be achieved by (a) life style management, (b) pharmacotherapy.

Life Style Management

1. Diet therapy:
    - Diet rich in fruits, vegetables, fibers and whole grain. Two servings of fish rich in omega-3 fatty acids per week.
    - Reducing sodium < 2300mg/day.
    - 45-65% of calories from carbohydrates 20-35% of daily calories from fat and (saturated fats to <10% of the daily calories) and 10-35% from protein.
    - Fibre intake: >50yrs - 38gm (men), 25gm (women) , <50yrs - 30gm (men) 21gm (women).

- Types of energy reduced diets: 1.Very low calorie diet - provides <800kcal/day, liquid meal fortified with minerals. It is recommended for morbidly obese and those who need rapid weight loss. 2. Low calorie diet – supplies 1200-1600kcal/day. It consists of traditional low calorie diet, prepackaged foods fortified with minerals. It provides nutritionally balanced low fat, low energy meal.[5555]

- Special nutrients – 1.Oat fibre, 2.Conjugated linoleic acids – decreasing lipid synthesis, increasing lipolysis 3. Whey protein – rich in essential, branched chain aminoacids.

1. **Physical activity:**

According to Physical activity 2008 guidelines, 150 minutes /week of moderate exercises or 75 minutes /week –vigorous ,aerobic exercises.

2. **Behaviour therapy:**

Stress management, avoid eating in front of television, introducing new diet/physical exercises.

3. **Medical management:**

1.Centrally and peripherally acting Anorexins,Endocannabinods and newer drugs Bupropion+naltrexone/bopropion+zonisamide,fenfluramine,

Lorcaserin,Orlistat,Phenttermine,Rominabsnt,Sibutramine and Tesofensine(under trial),

2. High and moderate intensity statin therapy.

High intensity statin therapy	Moderate intensity statin therapy
Daily dose lowers LDL-C on average by approximately ≥ 50%	Daily dose lowers LDL-C on average by approximately 30 - 50%
Atorvastatin – 40-80mg Rosuvastatin – 20-40mg	Atorvastatin – 10-20mg Rosuvastatin – 5-10mg Simvastatin – 20-40mg Pravastatin – 40-80mg Lovastatin – 40mg Pitavastatin – 2-4mg

TABLE 6: STATIN THERAPY

3. Non statin drugs like fibrates, nicotinic acid and omega-3 fatty acids. National Lipid Association (NLA) recommendation for management of lipidemia: Elevated ApoB containing lipoproteins (non HDL-C and LDL-C) are atherogenic cholesterol which focus on non HDL-C – (triglyceride management) in the non fasting state.

4. **Alternative medicine:**

   Green tea contains catechine, (antiobesogenic), hydrocitric acid present in Indian fruit Garcinia cambogia[61].

5. **Surgical management:**

   Indications: BMI >40kg/m^2 or BMI >35 kg/m^2 with comorbidities such as Hypertensive, hyperlipidemia severe arthrosis and DM[31].

   Surgical procedures for obesity seems to have potential for changing the current concepts of pathophysiology and management of metabolism.

BARIATRIC SURGERIES

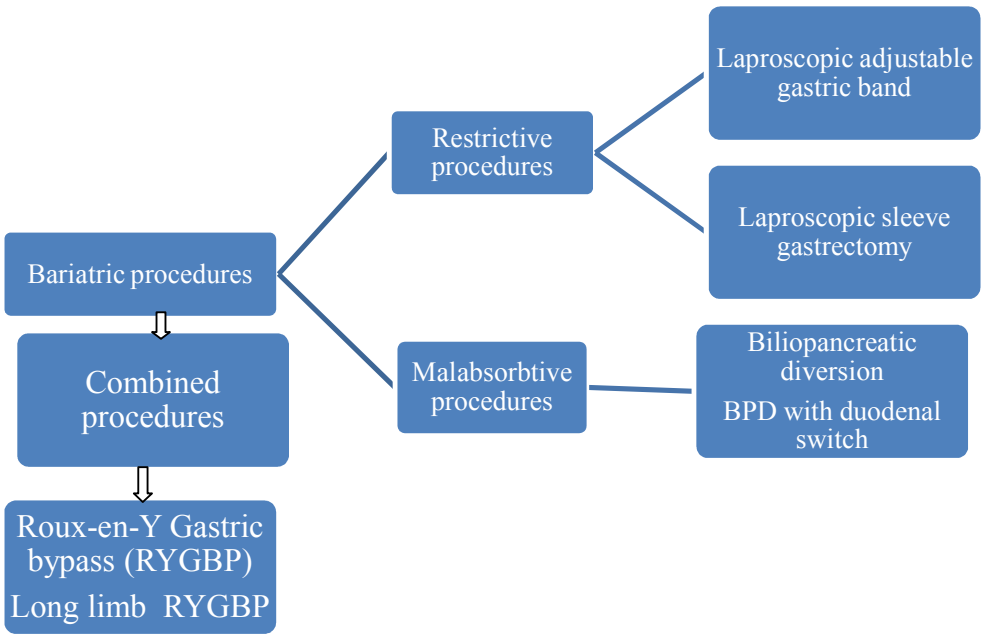


FIG 10: VARIOUS BARIATRIC SURGERIES

STUDIES

STUDY 1: TABLE 7

STUDIED BY	Ghosh A et al[32]	
TOPIC	Corelation between WHR and TC,FPG,FTGL	
STUDY POPULATION	130 bengalee	
WHR ≤0.95(CENTRALLY NON OBESE-CNO) N=47 Higher TC(p<0.05) FPG (p < 0.01) and FTG (p < 0.05)		WHR≥0.95(CENTRALLY OBESE-CO) N=83
RESULT	CO status had a significant effect (p < 0.05) on TC, VLDL-C, FPG and FTG.	

STUDY 2: TABLE 8

STUDIED BY	Asha Mahilmaran et al[33]
TOPIC	Corelation between BMI,WHR,TC with age
STUDY POPULATION	2318 people above 20 years at apollo hospital Chennai
Total cholesterol (TC) >200mg/dl was present in 36%, HDL,<40mg/dl (males), <50mg/dl (females) in 37%, LDL> 130mg/dl in 25%, triglyceride(TGL) > 150mg/dl in 41 % of the people. (p-0.001)	
RESULT	Significant increase in BMI, WHR after the age of 30 years (p,0.00I).

Mohan V et al[34] conducted a study on 1167 subjects residing in the residential areas of 2 industries (Indian Airlines and Integral Coach Factory) in Chennai in southern India. Diabetes was found in 11.9%; hypertension in 25.4%; dyslipdemia in 40.2%; hypertriglyceridemia in 28.3%; overweight (body mass index > or = 23 kg/m2) in 60.2%; and metabolic syndrome in 34.1%. Use of tobacco in any form was present in 22.9% of men and 0.5% of women.

STUDY 3: TABLE 9

STUDIED BY	Gupta R et al[35]
TOPIC	Prevalence (%) of cardiovascular risk factors in men and women had the habit of smoking/tobacco use
STUDY POPULATION	1091
Prevalence (%) of cardiovascular risk factors in men and women had the habit of smoking/tobacco use in 37.6 and 11.6, hypertension in 37.0 and 37.6%, overnight and obesity in 37.8 and 50.3, truncal obesity in 57.3 and 68.0, high cholesterol > or = 200 mg/dl in 37.4 and 45.8, high triglycerides > or = 150 mg/dl in 32.3 and 28.6 metabolic syndrome in 22.9 and 31.6 percent.	
RESULT	Significant increase in hypertension, obesity and metabolic syndrome among tobacco users.

STUDY 4: TABLE 10

STUDIED BY	Gupta R et al[36]
TOPIC	Prevalence of dyslipidemia
STUDY POPULATION	234 physicians
Prevalence of high total cholesterol levels > or = 200 mg/dl was in 91 (44.8%) males and 10 (32.3%) females. High LDL cholesterol level (> or = 100 mg/dl) was in 144 (70.9%) males and 22 (70.9%) females. LDL levels > or 130 mg/dl in 70 (34.5%) males and 9 (29.0%) female physicians. High triglyceride levels (> or = 200 mg/dl) were in 38 (18.7%) males and 4 (12.9%) female.	
RESULT	Significant prevalence of dyslipidemia among physicians

STUDY 5: TABLE 11

STUDIED BY	Misra A et al[37]
TOPIC	Correlation ofBMI, WHR with lipid profile
STUDY POPULATION	532 subjects
high waist-hip ratio (WHR) in 9.4% (95% CI 5.4-14.8) of males, 51.1% (95% CI 45.8-56.3) of the females	
RESULT	In both males and females BMI, WHR were positive predictors of biochemical parameters except for HDL cholesterol.

Arijit De et al[38] conduted a population-based study on 1005 persons. A total of 280 (27.9%) persons from the study population, were found to be hypertensive, of which 151(54%) persons were found to be dyslipidemic. Both males ans females hypertensives had significantly higher values of total cholesterol, TG, LDL-C as compared to control($p<0.001$).

STUDY 6: Table 12

STUDIED BY	Chadha DS et al[39]
TOPIC	Correlation BMI,WHR and lipid profile
STUDY POPULATION	From aug 2005 to Jan 2006 at the Air Force Central Medical Establishment, New Delhi.
Hypertriglyceridemia (36%) was the commonest abnormality noted followed by low HDL-C levels (18%). Lipid subfraction analysis revealed that cholesterol and LDL-C correlated to BMI($r=0.2, p=0.002$) and body fat% ($r=0.16, p=0.020$). in older men, BMI correlated positively with elevated cholesterol ($r=0.36, p<0.001$) while WHR correlated positively with triglycerides ($r=0.42, p=0.001$).	
RESULT	Combination of anthropometric variables predicts dyslipidemia better in asymptomatic healthy males than anyone particular variable.

STUDY 7: TABLE 13

STUDIED BY	AM Savant et al[40]
TOPIC	Correlation BMI,WHR and lipid profile
STUDY POPULATION	1085, a retrospective study in 2006 at P.D.Hinduja National Hospital and Medical research Center, Mumbai, India.
TC concentration 200 mg/dl, 38.7% were males and 23.3% were females. HDL-C was abnormally low in 64.2% males and 33.8% in females.	
RESULT	The increase of prevalence of hypercholesterolemia and hypertriglyceridemia was more prominent in 31-40 age group than in 30 age group.

STUDY 8: TABLE 14

STUDIED BY	Latheef SA et al[41]
TOPIC	Correlation hypertension,obesity and lipid profile
STUDY POPULATION	1519 urban population of Tirupati
central obesity- 1003 (66%) [male 320 (59.4) and females 683 (41.12%)], Low HDL cholesterol -709 (46.67%) [male 284 (52.69%) and females 425 (43.36%)], High triglyceride levels (>or= 150 mg/dl) 444 (29.2%)[(males 173 (32.09%) and females 272 (27.75%), high cholesterol levels (>or=200 mg/dl) [(males 127 (23.56%) and females 278 (28.36%) hypertension 396 (26.06%) [males 149 (27.64%) and females 247 (25.20%)].	
RESULT	Central obesity and hypertension are positively correlated to high TGL,TC and low HDL.

STUDY 9:TABLE 15

STUDIED BY	Chow CK et al[42]
TOPIC	Mean levels of total, LDL, HDL-cholesterol and triglycerides in population if Asian definitions are used.
STUDY POPULATION	4535 rural Andhra Pradesh
mean levels of total, LDL, HDL-cholesterol and triglycerides were 4.5 (4.4-4.6) mmol/L, 2.8 (2.7-2.9) mmol/L, 3.0 (3.0-3.1) mmol/L, 1.2 (1.16-1.22) mmol/L, 1.3 (1.2-1.4) mmol/L for women. 18.4% of men and 26.3% of women were overnight rising to 32.4% of men and 41.4% of women if 'Asian' definitions were used. Criteria for NCEP-ATPIII metabolic syndrome were met by 26.9% of men and 18.4% of women with figures of 32.5% and 23.9%, respectively, if 'Asian' waist cut-offs were substituted.	
RESULT	Asian weight cut offs gives more refined values for Indian population.

STUDY 10: TABLE 16

STUDIED BY	Gupta R et al[43]
TOPIC	Correlation between
STUDY POPULATION	1123 adults aged >20 years in an urban Indian population in Jaipur Heart watch study-2.
Obesity, body mass index > or = 27 kg / m2 was present in 135 males (24.5%) and 173 females (30.2%). Truncal obesity (waist :hip > 0.9 males, >0.8 females) was found in 316 males (57.4%) and 392 females (68.4%). The most comman dyslipidemia in both males and females was low HDL-cholesterol (<40 mg/dl: males 54.9%, females 54.2%). High total cholesterol levels of > or = 200mg/dl (males 37.4%, females 54.2%). High LDL-cholesterol levels of > or =130 mg/dl (males 37.0%, females 45.8%) and high levels of triglycerides > or = 150 mg/dl (males 32.3%, females 28.6%) were also seen in a significant number.	
RESULT	Most common dyslipidemia is low HDL, followed by high TGL and high TC and LDL.

Waisr JS et al[44] studied 2050 adult Asian Indian subjects residing in two metropolitan cities and found that hypertriglyceridemia (>150 mg/dl) in 20.2% and low levels of HDL-C (<40 in males; <50 mg/dl in females) in 55%.

Gupta A et al[45] studied 1091 urban Indian population aged 20 years and above, and it was found that among men and women high triglycerides in 32.1 and 28.6%, low HDL cholesterol in 54.9 and 90.2%; central obesity in 21.8 and 44.0% of individuals.

Gupta A et al[46] studied 1127 adults aged >20 years and found that among men and women obesity (BMI>or=25 kg/m2) in 303 (54.5%) and 350 (61.3%), truncal obesity (high WHR) in 339 (61.0%) and 310 (54.30%), hypertension in 322 (57.9%) and 279 (48.9%), high total cholesterol>or=200 mg/dl in 111 (32.6%) and 120 (39.5%), low HDL cholesterol<40mg/dl in 103 (30.3%) and 83 (27.3%), high triglycerides>or=150mg/dl in 146 (42.9%) and 132 (43.4%).

Gupta R et al[47] studied 1091 adults > 20 years in Indian urban population, central obesity (waist, men > 102 cm, women >88cm) was found in 116 (25.6%) and 246 (44.0%); low HDL cholesterol (men,40mg/dl, <1.0mmol/l), women<50 mg/dl, <1.3mmol/l) in 292 (54.9%) and 504 (90.2%); high triglycerides >/=150 mg/dl (>/=1.7mmol/dl) in 172 (32.3%) and 160 (28.6%).

Gupta R et al[48] examined 458 persons in Jaipur Heart watch study-3 and found that hypercholesterolemia (total cholesterol > or = 200mg/dl) in 75 (33.2) and 67 (28.9%), high triglycerides in 55 (24.3%) and 34 (14.7%), low high-density

lipoprotein cholesterol in 169 (74.8%) and 155 (66.8%),among men and women. There was significant linear relationship of body mass index with prevalence of hypercholesterolemia.

Gupta R et al[49] studied (in 1995)2212 subjects and 1123 subjects(in 2002) in an Indian urban population and found that , In men, high total cholesterol > or = 200mg/dl was in 24.6% vs. 37.4%, high LDL cholesterol > or = 130 mg/dl in 22.1 vs. 37.0%, high triglycerides > or = 150 mg/dl in 26.6 vs. 30.6% and low HDL cholesterol < 40 mg/dl in 43.2 vs. 54.9%; while in women these were in 22.5 vs. 43.1%, 28.6 vs. 45.1%, 28.6 vs. 28.7% and 45.9 vs. 54.2% respectively.

METHODOLOGY

Design and Data collection

This was a cross sectional study. The study was undertaken on the female subjects approaching the outpatient department of the Coimbatore Medical College Hospital, Coimbatore between September 2014 – August 2015. The study is proposed to be conducted after obtaining consent from the subjects.

Inclusion criteria

- Healthy female subjects without comorbid illness
- Age group between 25-60yrs

Exclusion criteria

- Diabetes mellitus
- Hypertension
- Ischemic heart disease
- Chronic renal disease
- Chronic liver disease
- Hypothyroidism
- Patients on lipid lowering drug

Information was collected about demographics (age, gender and occupation), anthropometric measurements (body Wt, Ht, BMI, WC, hip circumference and WHR), personal habits of dietary pattern (veg/mixed), alcohol intake(quantity), tobacco chewing and smoking and clinical parameters (BP, FBS, ECG) ,previous drug intake and F/H of diabetes, hypertension, ischemic heart disease). Blood collected by puncturing vein after 8-10 hours of overnight fasting.It was collected in red topped plain tubes for analysis. Routine urine analysis was carried out on the same day and ECG was taken for those who were found to be dyslipidemic.

Patients were interviewed as per the prepared Proforma and then complete clinical examination was done.

Blood Glucose and lipid subfraction Analysis

Blood samples are analysed by using automatic analyser. Blood glucose calculated by GOD-POD END POINT Trinder's method. TC was measured by DYNAMIC EXTENDED STABILITY CHOD-PAP METHOD, Triglyceride (TGL) concentrations were measured by TC was measured by DYNAMIC EXTENDED STABILITY CHOD-PAP METHOD, Triglyceride (TGL) concentrations were measured by DYNAMIC EXTENDED STABILITY WITH LIPID CLEARING AGENT GPO-TRINDER METHOD. IFCC approved enzymatic methods are used. LDL was estimated by Friedward's formula, VLDL was calculated by dividing TGL by Five.

Definitions and Cut off points of hyperlipidemia

According to NCEP- ATP III Guidelines ,serum lipid fractions are categorized into:1.Hypercholesterolemia defined as TC > 200mg/dl

2 LDL-C as > 100mg/dl,

3. Hypertriglyceridemia as TGL > 150mg/dl

According to ADA,.FBS > 126mg/dl /on anti diabetic drugs were considered as Diabetes Mellitus.

Statistical Analysis

Age, Anthropometric measurements, Serum Lipid subfraction and fasting glucose levels measured were calculated by using -mean $\pm$ SD. The data collcted were again categorized according to two age groups .By using Chi square test, Unpaired t test was applied in comparisons of independent and dependent proportions. A p value <0.05 was considered deemed significant. Results were expressed as percentages.

RESULTS

The study population was comprised of 100 asymptomatic females aged between 25-40years.

TABLE 17: DEMOGRAPHIC AND CLINICAL CHARACTERISTICS OF THE STUDY POPULATION

Parameter	Means	SD±	P Value
Age	30.76	4.73	-
Weight	59.43	7.67	>0.05
Height	158.23	5.88	>0.05
BMI	23.69	2.69	<0.01
WC	78.82	2.71	<0.01
WHR	0.87	0.05	<0.05
Systolic BP	117.70	9.23	-
Diastolic BP	78.08	5.90	-

Unpaired t test was applied

$P < 0.05$ is significant

In 100 asymptomatic females studied, the mean age of the females was 30.76 ± 4.73. The mean weight was 59.43 ± 7.67. Mean BMI found was 23.69 ± 2.69kg/m^2. Mean waist circumference were 78.82 ± 2.71 cms. The mean waist hip ratio were 0.87 ± 0.05, whereas the mean systolic and diastolic BP was 117.70± 9.23mmHg and 78.08 ± 5.90mmHg respectively. There was significant difference between means of BMI (P value <0.01), WC (P value <0.01), and WHR (P value <0.05).

TABLE 18: BIOCHEMICAL CHARACTERISTICS OF THE STUDY POPULATION

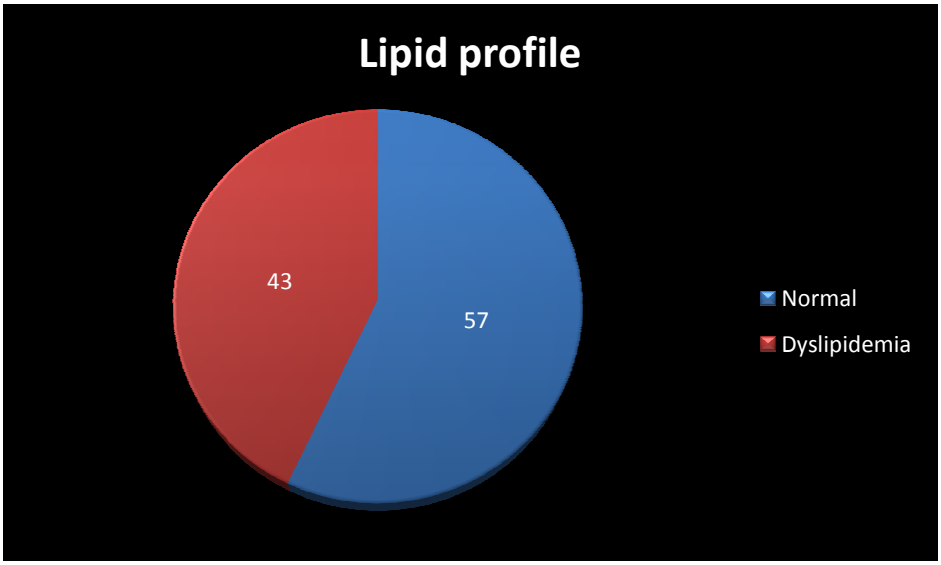
Parameter	Means	SD±	P Value
TC	169.54	24.33	>0.05
TGL	134.27	19.73	>0.05
LDL	103.85	24.45	>0.05
HDL	39.85	5.91	>0.05
VLDL	24.52	14.16	0.02
FBS	98.99	12.04	>0.05

Mean cholesterol found were 169.54 ± 24.33mg/dl., whereas the mean triglycerides were 134.27 ± 19.73mg/dl. The mean LDL,HDL,VLDL were 103.85 ± 24.45, 39.85 ± 5.91 and 24.52 ± 14.16mg/dl. The mean fasting blood sugar was 98.99 ± 12.04. There was significant difference between means of VLDL (P value 0.02).

TABLE 19: DISTRIBUTION OF LIPID PROFILE AMONG ASYMPTOMATIC FEMALES

Lipid profile	No
Normal	57
Dyslipidemia	43
Total	100

FIG 11: DISTRIBUTION OF LIPID PROFILE



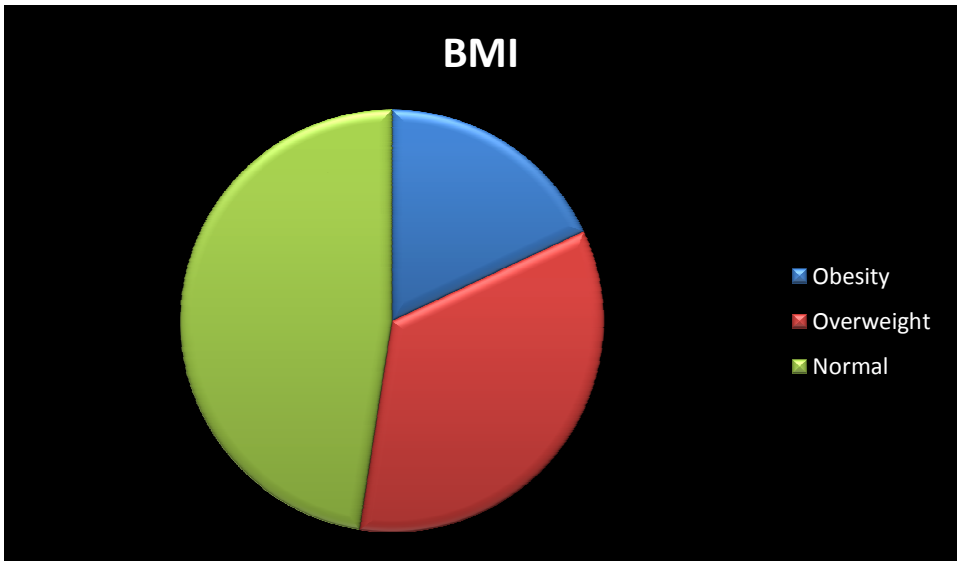
Among 100 asymptomatic females, 43% were found to be dyslipidemic.

Remaining 57% were normolipidemic.

TABLE 20: DISTRIBUTION OF BMI AMONG ASYMPTOMATIC FEMALES

BMI	≤30Yrs	31 to 40Yrs
Obesity	11	15
Overweight	21	12
Normal	29	12

FIG 12: DISTRIBUTION OF BMI

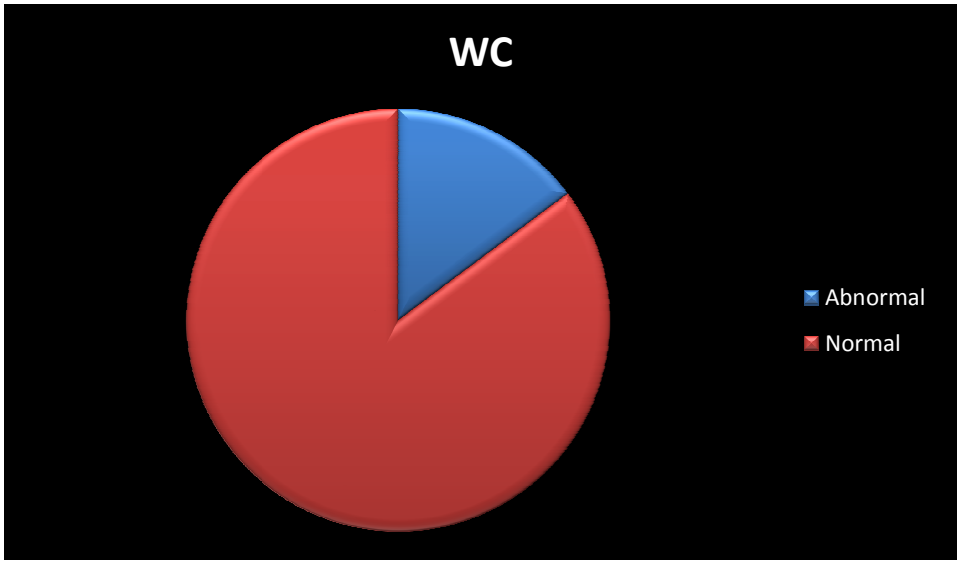


Among 100 asymptomatic females 41 were found to be normal BMI, 26 were obese, 33 were found to be over weight.

TABLE 21: DISTRIBUTION OF WC AMONG ASYMPTOMATIC FEMALES

WC	<30Yrs	31 to 40Yrs
Abnormal	9	15
Normal	52	24

FIG 13: DISTRIBUTION OF WC

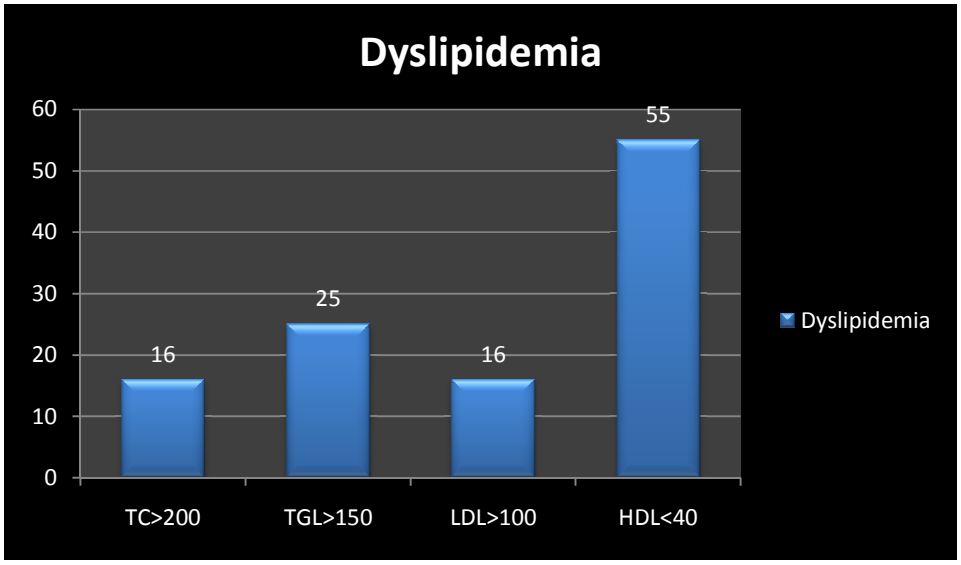


Among 100 asymptomatic females, 76 were found to be normal Waist Circumference, 24 were found to be abnormal.

TABLE 22: PATTERN OF DYSLIPIDEMIA IN ASYMPTOMATIC FEMALES

Parameter	No	P value
TC>200	16	>0.05
TGL>150	25	>0.05
LDL>100	16	>0.05
HDL<40	55	>0.05

FIG 14: PATTERN OF DYSLIPIDEMIA



In 100 asymptomatic females studied, the HDL was <40mg/dl is 55, followed by TGL >150mg /dl in 25, the TC >200mg/dl and LDL >100mg/dl were 16 each.

TABLE 23 : DISTRIBUTION OF BMI AND DYSLIPIDEMIA

		DYSLIPEDEMIA				TOTAL	
		YES		NO		No.	%
		No.	%	No.	%		
BMI	Obese (>25)	24	92.3	2	7.7	26	100.0
	Over wt (22.5-25)	7	21.2	26	78.8	33	100.0
	Normal (<=22.5)	13	31.7	28	68.3	41	100.0
TOTAL		44	44.0	56	56.0	100	100.0

Dyslipidemia was present in 92.3% of obese persons, 21.2% of overweight persons and 31.7% of normal BMI persons. There was significant correlation between BMI and abnormal lipid profile.(P value <0.01)

TABLE 24 :DISTRIBUTION OF WC AND DYSLIPIDEMIA

		DYSLIPEDEMIA				TOTAL	
		YES		NO		No.	%
		No.	%	No.	%		
WC	Above Normal (>80)	22	91.7	2	8.3	24	100.0
	Normal (<=80)	22	28.9	54	71.1	76	100.0
TOTAL		44	44.0	56	56.0	100	100.0

Abnormal Waist circumference was seen in 91.7% of the study population,it has significance with dyslipidemia. (P value<0.01)

TABLE 25: DISTRIBUTION OF DYSLIPIDEMIA

BASED ON AGE

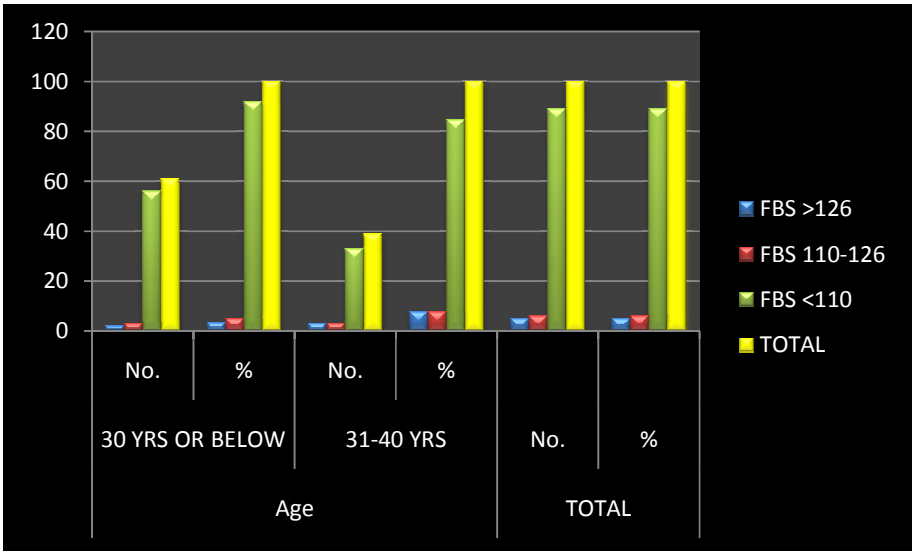
Parameter	Age			
	<30Yrs		31 to 40Yrs	
	No	%	No	%
TC	8	13.1	8	13.1
TGL	13	21.3	12	30.8
LDL	8	13.1	8	13.1
HDL	31	50.8	24	61.5

Among 100 asymptomatic females, low HDL and high TGL were found to be higher in 31-40years age group than below 30years age group. 61.5% were found to be low HDL in 31-40years age group, 50.8% were below 30years.

TABLE 26: INCIDENCE OF DIABETES AND IMPAIRED FASTING GLUCOSE IN ASYMPTOMATIC FEMALES

		Age				TOTAL	
		30 YRS OR BELOW		31-40 YRS		No.	%
		No.	%	No.	%		
FBS	>126	2	3.3	3	7.7	5	5.0
	110-126	3	4.9	3	7.7	6	6.0
	<110	56	91.8	33	84.6	89	89.0
TOTAL		61	100.0	39	100.0	100	100.0

FIG 15: INCIDENCE OF DIABETES AND IMPAIRED FASTING GLUCOSE



Out of 100 asymptomatic females studied, Diabetes was found in 5 percent. Among them 3.3 percent were below 30 years,7.7 percent were in 31-40 year age group. However there was no statistical significance (P value >0.05).

TABLE 27: AGE SPECIFIC DISTRIBUTION OF CORONARY RISK FACTORS AMONG STUDY POPULATION

Characteristics	Age group		P Value
	≤30Yrs (61)	31 to 40Yrs (39)	
Hypercholesterolemia (>200mg/dl)	8(13.1)	8(20.5)	>0.05
Hypertriglyceridemia (>150mg/dl)	13(21.3)	12(30.8)	>0.05
High LDL (>100mg/dl)	8(13.1)	8(20.5)	>0.05
Low HDL (<40mg/dl)	31(50.8)	24(61.5)	>0.05
High FBS (≥126mg/dl)	2(3.3)	3(7.7)	>0.05
High WC (≥80cm)	9(14.8)	15(38.5)	<0.05
High W/H Ratio (≥0.80cm)	61(100)	39(100)	-
High BMI (≥25kg/m^2)	11(18)	15(38.5)	>0.05
High BP (≥130/85mmHg)	6(9.8)	8(20.5)	>0.05

There was 61 asymptomatic females in the age group of $\leq$ 30years compared with 39 in 31-40years age group. The low HDL (<40mg/dl) was high among 31-40years age group (P value >0.05). WC was also higher in 31-40years age group. (P value <0.05). High BMI was found in age group 31-40years (P value >0.05). High BP was found in age group 31-40years (P value >0.05). WC has statistical significance (P value <0.05) among 31-40years age group.

TABLE 28: CORONARY RISK FACTORS AND HABITS

Parameter	Tobacco chewer (n=26)	Non tobacco chewer(n=74)	P Value
TC>200	9	7	<0.01
TGL>150	16	9	<0.01
LDL>100	9	7	<0.01
HDL<40	6	49	<0.01
WC≥80cm	15	9	<0.01
WHR>0.7	26	74	-
BMI≥25	17	9	<0.01

Parameter	Alcoholic n=8	Non alcoholic n=92	P Value
TC>200	4	12	<0.05
TGL>150	6	19	<0.01
LDL>100	4	12	<0.05
HDL<40	1	54	<0.05
WC≥80cm	8	16	<0.01
WHR>0.7	8	92	-
BMI≥25	6	20	<0.01

Parameter	Veg n=16	Mixed n=84	P Value
TC>200	7	9	<0.01
TGL>150	11	14	<0.01
LDL>100	7	9	<0.01
HDL<40	4	51	<0.01
WC≥80cm	10	14	<0.01
WHR>0.7	16	84	-
BMI≥25	11	15	<0.01

In 100 asympyomatic females,26 were chewing tobacco,8 were alcoholic,16 were vegetarians.Dyslipidemia was found to be significant in tobacco chewers (P value <0.01) and alcoholic (P value <0.05) .BMI and WC were higher in tobacco chewer (P value <0.01).

WC was higher in those who had vegetarian diet (P value <0.01) .BMI and WC were higher in alcoholic (P value <0.01).

TABLE 29: PEARSON CORRELATION COEFFICIENT ANALYSIS OF VARIABLES OBESITY AND DYSLIPIDEMIA

		DYSLIPIDEMIA
BMI	Pearson Correlation	0.393**
WC	Pearson Correlation	0.549**
WHR	Pearson Correlation	0.260*

**.Significance at 0.01 level.

*Significance at 0.05 level.

There was significant correlation between BMI, WC and WHR. Among them, BMI and Waist Circumference were significant at P value <0.01.

TABLE 30: PEARSON CORRELATION COEFFICIENT ANALYSIS OF VARIABLES OBESITY AND LIPID PROFILE

		TC	TGL	LDL	HDL
BMI	Pearson Correlation	.396**	.597**	.421**	-.330**
WC	Pearson Correlation	.646**	.664**	.703**	-.529**
WHR	Pearson Correlation	.204*	.369**	.236*	-.268**

** Significance at 0.01 level.

* Significance at 0.05 level.

Pearson correlation coefficient analysis showed significant correlation between BMI with TGL($P<0.01$), LDL($P<0.01$), HDL($P<0.01$). Waist Circumference had significant correlation with TC($P<0.01$), TGL($P<0.01$), LDL($P<0.01$).

TABLE 31: PEARSON CORRELATION COEFFICIENT ANALYSIS OF LIPID SUBFRACTIONS TO VARIABLES OF OBESITY IN VARIOUS AGE GROUPS

Variables	≤30yrs	31-40yrs
Total cholesterol		
BMI	0.323*	0.451**
WC	0.734**	0.551**
WHR	0.196	0.161
Triglycerides		
BMI	0.530**	0.690**
WC	0.789**	0.690**
WHR	0.349**	0.382*
LDL		
BMI	0.349**	0.482**
WC	0.789**	0.626**
WHR	0.247	0.185
HDL		
BMI	-0.212	-0.606**
WC	-0.430**	-0.743**
WHR	-0.255*	-0.339*

There was significant correlation between BMI and WC with total cholesterol ($r = 0.323$ and $r = 0.734$, $p < 0.05$, triglycerides ($r = 0.530$ and $r = 0.789$, $p < 0.01$) in $\leq$30yrs age group. BMI, WC were significantly correlated with HDL ($r = 0.606$, $p < 0.05$, $r = 0.743$, $p < 0.05$) in 31-41yrs age group. TGL and LDL also were significantly correlated with BMI, WC and WHR in both age groups.

DISCUSSION

In our study ,a step towards evaluating the blood level of lipids and glucose in asymptomatic females in urban population and identifying these coronary risk factors by anthropometric variables. The study identifies the pattern of dyslipidemia such hypercholesterolemia, hypertriglyceridemia and abnormally high Low Density Lipoprotein cholesterol(LDL) and low High Density Lipoprotein cholesterol (HDL) levels in all age groups, which are well known risk factors for CardioVascular Diseases.

Overall prevalence of dyslipidemia was ranged from 10-73% in various studies[37].In our study43% of asymptomatic females were found to be dyslipidemic.Our results are consistent with the previous cross sectional study conducted among Sudanese women in Khartoum state[25] and across sectional study conducted at Tirupathi[41] wherein increased prevalence of dyslipidemia in young females was found to be one of the major contributor of Coronary Artery Disease(CAD).

Our study which showed that elevated lipid levels were prominent in 31-40year age group as compared to ≤30 years,which means the risk of dyslipidemia increases as the age advances .In our study, fasting blood sugar found to be more in 31-40 year age group and of these 7.7%were found to be newly diagnosed Diebetes Mellitus.The remaining subjects with impaired fasting blood sugar levels (6%) are on their way to become Diabetic in future, which is an important risk factor for CAD.Enas et al,in Coronary artery disease in Indians (CADI) study reports the prevalence of diabetes mellitus to be three to six times higher among south Asians than Europeans, Americans and other Asians[45]. In our study,Dyslipidemia was found significant among tobacco chewers and alcoholics (P value<0.01).This is consistence with the study done by Gupta BK et al[50].

In our study , the most common dyslipidemic pattern observed was low HDL cholesterol(61.5%). These finding is consistant with Jaipur Heart Watch-2[51],Jaipur Heart Watch-3[47] studies and cross sectional study which was conducted at Tirupati[41].In our study,low HDL cholesterol was not differs significantly between the age group ≤30 years(50.84%) and 31-40years(61.5%)[37].HDL cholesterol particles are mainly involved in reverse cholesterol transport,leading to antiatherogenic effect.HDL is more specific in prevention and removal of cholesterol deposits within the arterial

wall[52].Low HDL cholesterol levels are stronger predictor of premature and severe Coronary Artery Disease(CAD) and Stroke and its recurrence.[54]Increased prevalence of low HDL cholesterol was reported in earlier study by Enas et al,who found that only 5% of Asian Indian women had optimal HDL levels[53].

In the process of Atherosclerosis,high LDL cholesterol and its oxidation has been recognized as primary risk factor for Coronary Artery Disease by National Cholesterol Education Programme(NCEP) Adult Treatment Plan(ATP)-III.[53]In our study,low LDL cholesterol was found 20.5% in31-40 years age group,13.1% among ≤30 years because of unhealthy food habits and life style modifications.

In our study,the second common lipid subfraction contributing to dyslipidemia was hypertriglyceridemia which was 30.8% in the age group of 31-40 years,21.3% among ≤30 years.The hypertriglyceridemia, hypercholesterolemia and high LDL is a major risk factor of concern in the age group of 31-40 years. When compared to western population ,Indians had relatively low serum cholesterol predispose them to CAD.[55]In Chennai based hospital study,the threshold for the TC levels posses the risk for CAD was<200mg/dl and 75% of Myocardial Infarction(MI) patients had less than 200mg/dl.[40] Diet rich in fat and high calorie intake ,reduced physical

activity would be the major culprits of Dyslipidemia in our population.Our dietary pattern are rich in saturated fats[56] and also involves destruction of nutrients like folate by overcooking and frying.Reuse of the same cooking oil for frying,refrying leading to transfattyacid formation which probably contributes to increase risk of Dyslipidemia in our population.[40]In Canadian study, influence of diet on Dyslipidemia was studied by utilizing 3 groups:a control group, a group with only dietary modifications and a group that was started on statin. After 4 weeks, lipid profile was repeated and compared.A drastic reduction in lipid levels was noted in both diet and statin group than control group. However, between them not much variation in lipid levels[57].Both of them have same effect, and dietary intervention is a more viable option.

In our study, Body Mass Index (BMI) ,Waist Circumference(WC) and Waist to Hip Ratio(WHR) correlated with Dyslipidemia in female population.Further correlation analysis between BMI and lipid subfraction revealed that BMI showed significant correlation with TGL($P<0.01$), LDL($P<0.01$), HDL($P<0.01$). Waist Circumference had significant correlation with TC($P<0.01$), TGL($P<0.01$), LDL($P<0.01$).WHR also had significant correlation with TC ($P< 0.05$), LDL ($P<0.05$)and TG ($P<0.01$)These measures were consistent with studies conducted by Chada

DS et al[39],Misra A et al[37,58].BMI was widely used as an indicator of obesity ;it has some limitations because it dependent on race,age and body fat percentage.Asians had low BMI and high body fat .Abdominal obesity has been significantly associated with CVD risk which has been measured by anthropometrics indices like WC and WHR especially in females [59,60]. In Nurses Health study conducted by Rexrode KM et al,they identified WC and WHR both are independent risk factor of CAD in women.[39]

Dyslipidemic persons are more frequently 'centrally obese''. [37]especially females. In our study, BMI,WC and WHR all are correlated with lipid subfraction analysis. None of the anthropometric measures when used alone was able to predict dyslipidemia better. In our country, physicians should always consider combination of anthropometric parameters like WC and WHR in addition to BMI to assess the cardiometabolic health.

SUMMARY

- Our study was conducted at CMCH Coimbatore from September 2014 to August 2015.
- The study population was comprised of 100 asymptomatic (resembling healthy) females came for routine screening in master health checkup.
- Overall 43% were found to be dyslipidemic, 57%were normolipidemic.
- Low HDL cholesterol <40mg/dl was the most comman dyslipidemic pattern found in 55 persons followed by TG>150, in 25 persons and LDL>100, TC>200 found in 16 persons.
- Lipid subfraction analysis showed HDL was found in 61.5% in ≤31-40 years age group, 50.8% ≤30 years age group.
- 7.7% were found to be diabetic in 31-40years age group, 3.3% ≤30 years age group, impaired blood glucose was found in 6% of the total study population.
- Waist Circumference (P value <0.05) was significantly higher in 31-40 years age group (38.5%) and it was found 14.8% ≤30 years age group.

- Hyperlipidemia was found significantly higher (P value <0.01) among tobacco chewers and mixed diet persons (P value <0.01).
- BMI was higher in tobacco chewers (P value <0.01), WC (P value <0.01) and those who had mixed diet (P value <0.01) WC was significantly correlated with LDL
- BMI was significantly correlated with total cholesterol (r = 0.451, P <0.01), TGL (r = 0.690, P<0.01), HDL (r = 0.606, P<0.01), LDL (r = 0.482, P<0.01).
- WC was significantly correlated with total cholesterol (r = 0.734, P<0.01, TGL (r = 0.789, P<0.01),LDL-C (r=0.789, P<0.01) and with HDL(r = 0.430, P<0.01)
- WHR was significantly correlated with TGL (r = 0.382, P<0.05),HDL (r=0.339,P <0.05).
- BMI was correlated with HDL(P<0.05) in 31-40years age group.
- Combination of anthropometric measurements predicted dyslipidemia better in asymptomatic adults than anyone particular variable.

LIMITATIONS

- ✓ Genetic and secondary causes producing dyslipidemia were not ruled out.
- ✓ Lipoprotein estimation was not done.
- ✓ LDL-C was not estimated separated in those who had high triglyceride level
- ✓ Sample size was less due to time constraint.

CONCLUSION

- ✓ Dyslipidemia is more prevalent in young asymptomatic females being more common among 31-40yrs age group
- ✓ Low HDL Cholesterol was the most common dyslipidemic pattern being common in both <30 yrs and 31-40 yrs age group.
- ✓ BMI ,WC and WHR all of them have significant association with lipid subfraction analysis.
- ✓ Combination of anthropometric measurements predicted dyslipidemia better in asymptomatic females than single one variable.
- ✓ Therapeutic life style changes which is the main component in primary and secondary prevention of dyslipidemia.
- ✓ There is increasing trend of obesity and dyslipidemia in asymptomatic young females probably because of sedentary life styles.
- ✓ Early preventive measures needed to control overweight and prevent the future complications and disabilities.
- ✓ Health education about nutritious diet to be started in childhood will change the society into the healthy way.

BIBLIOGRAPHY

1. Chaturvedi V.Bhargava B.Health care Delivery for coronary heart disease in india - Wheare are we headed. Am heart hosp j 2007:5:32-37.

2. Hyman Levy, M.D. ; Ernst. P.Boas, M.D Coronary Artery Disease in Women. JAMA.1936; 107(2): 97-102. Doi:10.1001/jama. 1936. 02770280007002.

3. Findlay,IN,Cunningham.D,Dargie HJ.The RightS of Women.Br .Heart.J 1994;71:40/-3

4. Indrayan A.Forecasting vascular disease cases and associated mortality in india. Reports of the National Commoission on Macroconomics and Heath. Ministry of Health and family welfare. India 2005.

5. Executive summary of the third report of the National Cholesterol Education Programe (NCEP) Expert Panel on Detection. Evaluation, and treatment of high Blood Cholesterol in (Adult Treatment Panel III) [Special Communication].JAMA 2001:285:2486-2947.

6. Murabito JM[1], Evans JC, Larson MG, Levy D. An investigation of differences in outcome between the sexes according to initial Coronary disease presentation. Circulation 1993 ^{Dec}; $88^{(6)}$:2548-55.

7. Reeder BA, Senthiselvan A.Despres JP, Angel A.Liu L. Wand H.Rabkin SW. The association of cardiovascular disease risk factors with abdominal obesity in Canada. Canadian Heart health surveys Research Group. CMAJ. 1997:157:S39-S45.

8. Shettley SM, Marhall JA, Baxter J, Hamman RF. Waist-hip-ratio measurement location influences associations with measures of glucose and lipid metabolism. The san luis Valley Diabets Study. Ann Epidemiol 1993:3:295-299.

9. Bjorntorp P.reginaol patters of fat distribution. Ann int med.1985:103:994-995. Scidell JC, Cigolini M, Charzewska J, Ellsinger BM, di Base G. Fat Distribution in Eropean women : a comparision of anthropometric measurements in relation to cardiovascular risk factors. Int J Epidmiol, 1900:19:303-308.

Pouliot MC, Despres JP, Lemiecux S.Moorjani S.Bouchard C.Trembley A, nadeau A.Lupien PJ, Waist circumference and abdominal sagittal diameter: best simple anthropometric indexes of abdominal visceral adipose tissue accumulation and related cardiovascular risk in men and women. Am J cardiol. 1994:73:460-468.

10. Houmard JA, Wheeler Ws, McCammaon MR.Holbert D.Israel RG, Barakat HA, Wells JM, Truitt N,Hamad SF. An evaluation of waist to hip ratio measurement methods in relation to lipid and carbohydrate metabolism in men. Int J obes. 1991 :15:181-188.

Reader BA, Liu L. Horlick L. Selective Screening for dyslipidemia in a Canadian population. J Clin Epidemiol, 1996:49:217-222. doi : 10.1016/0895-4356(95)00063-1

11. Paccaud F, Schluter-Fasmeyer V, Wietlisbach V, Bovet P. Dyslipidemia and abkominal obesity: An assessment in three general populations. J Clin Epidemiol. 2000;53:393-400. doi: 10.1016/S0895-4356(99)00184-5.

12. Reader BA, Liu L. Horlick L. Selective Screening for dyslipidemia in a Canadian population. J Clin Epidemiol, 1996:49:217-222. doi : 10.1016/0895-4356(95)00063-1.

13. Thomas A. Gaziano, J. Michael Gaziano, Epidemiology of Cardiovascular disease, Harrison's Principles of Internal Medicine,The Mc Graw-Hill Companies,USA, 19th edition 2010,chapter-6e.

14. Anonymous. Expert Panel. Summary of the second report of the National Cholesterol Education Program (NCEP) expert panel on detection, evaluation, and treatment of high blood cholesterol in adults (adult treatment panel II). JAMA 1993;269:3015-3023.

15. Rodondi KM: Hyperlipidemia, in Herfindal ET, Gourley DR (eds): Textbook of Therapeutics: Drug and Disease Management. Baltimore, Williams & Wilkins; 1996: 387-403.

16. Farnier M, Davignon J: Current and future treatment of hyperlipidemia: the role of statins. American Journal of Cardiology 1998;82:3J-10J.

17. Anonymous. Expert Panel. Summary of the second report of the National Cholesterol Education Program (NCEP) expert panel on detection, evaluation, and treatment of high blood cholesterol in adults (adult treatment panel II). JAMA 1993;269:3015-3023.

18. Rubenfire M, Coletti AT, Mosca L: Treatment Strategies for Management of Serum Lipids: Lessons Learned From Lipid Metaboilism, Recent Clinical Trials, and Experience With the HMG CoA Reductase Inhibitors. Progress in Cardiovascular Diseases 1998;41:95-116.

19. Schaefer E, Levy R: Pathogenesis and management of lipoprotein disorders. New England Journal of Medicine 1985;312:1300-1310.

20. Ginsberg HN: Lipoprotein metabolism and its relationship to atherosclerosis. Medical Clinics of North America 1994;78:1-20.

21. Israel MK, McKenney JM: Hyperlipidemias, in Carter B, Angaran D, Lake KD, et al (eds): Pharmacotherapy Self-Assessment Program. Kansas City, American College of Clinical Pharmacy;1995:65-94.

22. McKenney JM: Dyslipidemias, in Lloyd YY, Koka-Kimble MA (eds): Applied Therapeutics: The Clinical Use of Drugs. Vancouver, Applied Therapeutics Inc; 1995: 1-26.

23. Israel MK, McKenney JM: Hyperlipidemias, In Carter B, Angaran D, Sisca T (eds): Pharmacotherapy Self-Assessment Program. Kanasas City, American College of Clinical Pharmacy; 1991: 27-47.

24. Rodondi N, den Elzen WP, Bauer DC, et al. Subclinical thyroid dysfunction, cardiac function and the risk of heart failure. The Cardiovascular Health study. J AM Coll cardiol 2008; 52:1152-9.

25. Sawant AM, Shetty D, Mankeshwar R, Ashavaid T F. Prevalence of Dyslipidemia in Young Adult Indian Population. JAPI 2008;56: 99-102

26. Andrea Dunaif, Women's health.Harrison's Principles of Internal Medicine,The Mc Graw-Hill Companies,USA, 19th edition 2010,chapter-6e.

27. Gupta.R.Diabetic dyslipidemia,apolipoproteins and vascular risk.Diab.Metab.Syndr:Clin Res Rev.2007;1:49-51.

28. SB.Heymsfield et al.Weight management using a meal replacement strategy:Meta and pooling analysis from six studies, International Journal of besity.2003;23:537-49.

29. Robert F.Kushner,Evaluation and Management Obesity,Longo et al. Harrison's Principles of Internal Medicine,The Mc Graw-Hill Companies,USA, 18th edition.2012:629-36.

30. M.Boschmann et al. The effects of epigallocatechins-3 gallate on thermogenesis and fat oxidation in obese men,a pilot study.The Journal of the college of Nutrition.2007

31. National Heart,Lung, And Blood Institute,North AmericanAssociation for the study of Obesity:The Practical Guide,IdentificationEvaluation,And Treatment of Overweight and Obesity in Adults.

32. Ghosh A, Bose K, Chakravarti S, Chaudhuri AB, Chattopadhyay J, Dasgupta G, Sengupta S. Central obesity and coronary risk factors. J R Soc promot Health 2004 Mar;124(2):86-90.

33. Mahilmaran A, Sowjanya S, Chandrasekaran, Sathyamurthy I, Balasubramanium U. Prevalence of Coronary Risk Factors in Asymptomatic Subjects Attending Preventive Health Check up. Indian Heart J 2005;57:543-554.

34. Mohan B, Deepa M, Farooq S, Prabhakaran D, Reddy KS. Surveillance for risk factors of cardiovascular disease among an industrial population in sothern India. Natl Med J India 2008 Jan-Feb;21(1):8-13.

35. Gupta R, Sarna M, Thanvi J, Sharma V, Gupta VP. Fasting glucose and cardio vascular risk factors in an urban population. JAPI 2007 Oct;55:705-9.

36. Gupta A, Gupta R, Lal B, Singh AK, Kothari K. Prevalence of coronary risk factors among Indian physicians. JAPI 2001 Dec;49:1148-52.

37. Misra A, Pandey RM, Devi JR, Sharma R, Vikram NK, Khanna N, High prevalence of diabetes, obesity, and dyslipidemia in urban slum population in northern India. Int J Obes Relat Metab Disord 2001 No;25(11):1722-9.

38. Arijit De, Kotokey RK, Hazarika S, Kalita BC, Khanwar RK.Profile of Lipid Abnormalities in Hypertensive Urban Population of Dibrugarh District of Upper Assam. Indian Heart J 2005;57: 543-554.

39. Chadha DS, Singh G, Kharbanda P, Vasdev V, Ganjoo RK. Anthropomametric correlation of lipid profile in healthy aviators. IJASM 2006; 50(2):32-37.

40. Sawant AM, Shetty D, Mankeshwar R, Ashavaid T F. Prevalence of Dyslipidemia in Young Adult Indian Population. JAPI 2008;56: 99-102

41. Latheef SA, Subramanyam G. Prevalence of coronary artery disease and coronary risk factors in an urban population of Tirupati. Indian Heart J 2007 Mar-Apr;59(2):157-64.

42. Chow CK, Naidu S, Raju K, Raju R, Joshi R, Sullivan D, Celermajer DS, Neal BC. Significant lipid, adiposity and metabolic abnormalities amongst 4535 Indians from a developing region of rural Andhra Pradesh. Atherosclerosis 2008 Feb; 196(2): 943-52. E-pub 2007 Apr 27.

43. Gupta R, Sarna M, Thanvi J, Sharma V, Gupta VP. Fasting glucose and cardio vascular risk factors in an urban population. JAPI 2007 Oct;55:705-9.

44. Wasir JS, Misra A, Vikram NK, Pandey RM, Gupta R. Comparison of definitionsof the metabolic syndrome in adult Asian Indians. JAPI 2008;56:158-164.

45. Gupta A, Gupta R, Lal B, Singh AK, Kothari K. Prevalence of coronary risk factors among Indian physicians. JAPI 2001 Dec;49:1148-52.

46. Gupta A, Gupta R, Sarna M, Rastogi S, Gupta VP, Korhari K. Prevalence of diabetes, impaired fasting glucose and insulin resistance syndrome in an urban Indian population. Diabetes Res Clin Pract. 2003 Jul;61(1):69-76.

47. Gupta R, Sarna M, Thanvi J, Rastogi P, Kaul V, Gupta VP. High prevalence of multiple coronary risk factors in Punjabi Bhatia community: Jaipur Heart Watch-3. Indian Heart J 2004 Nov-Dec;56(6):646-52.

48. Gupta R, Kaul V, Bhagat N, Agrawal M, Gupta VP, Misra A, Vikram NK. Trends in prevalence of coronary risk factors in an urban Indian population: Jaipur Heart Watch-4. Indian Heart J 2007 Jul-Aug;59(4):346-53.

49. Gupta R, Deedwania PC, Gupta A, Rastogi S, Panwar RB, Kothari K. Prevalence of metabolic syndrome in an Indian urban population. Int J cardiol 2004 No;97(2): 257-61.

50. Gupta BK, Kaushik A, Panwar RB, Chaddha VS, Nayak KC, Singh VB, Gupta R, Raja S. Cardiovascular risk factor in tobacco-chewers: a controlled study.JAPI 2007 Jan;55.27-31.

51. Gupta, Gupta VP, Sarna M, Bhatnagar S, Thanvi J. Sharma V, Singh AK, Gupta JB, Kaul V. Prevalence of coronary heart disease and risk factors in an urban Indian population: Jaipur Heart Watch-2. Indian Hear J 2002 Jan-Feb;54(1):59-66.

52. Peggy K. Han, PharmD, William C. Gong. PharmD, and Mark A. Gill, PharmD. Current Pathophysiology, Classification, Diagnosis, and Treatment Options of Dyslipidemia.

53. Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III) final report [special communication]. Circulation 2002;106: 3143-3421.

54. Mora S, Szklo M, and Otwos JD. LDL particle subclasses, LDL particle size, and carotid atherosclerosis in the Multiethnic Study of Atherosclerosis (MESA). Atherosclerosis 2007;192:211-17.

55. Kumar S, Roy S. Tropical Heart Diseas in India. In: Mantosh Panja Editors Dyslipdemia in Indians. Mumbai. Indian College of Physicians 2005;109-18.

56. Soysal A, Demiral Y. Soysal D, Ucku R, Koseoglu M, Aksakoglu G. The prevalence of metabolic syndrome among ypund adults in lzmir, Turkey. Annadolu Kardiyou Derg 2005;5:196-201.

57. Enas AE, Senthilkumar A, Hancy C, and Marc AB. Prudent diet and preventive nutrition from pediatreics to geriatrics: current knowledge and practical recommendations. Indian Heart J 2003;55:310-38.

58. Hu D, Hannah J, Gray S. Effects of Obesity and Body Fat Distribution on Lipids and Lipoproteins in Nondiabetic American Indians: The strong heart study. Obesity Research 2000;8:411-21.

59. Walton C, Lees B, Crook D, Worthington M, Godsland IF, Stevenson JC, Body fat distribution, rather than overall adiposity, influences serum lipids and lipoproteins in healthy men independently of age. Am J Med 1995;16:125-33.

60. Chumlea WC, Baumgartner RN, Garry PJ, Rhyne RL, Nicholson C, Wayne S, Fat distribution and blood lipids in a sample of healthy elderly people. J Obes Relat Metab Disord 1992;16:125-33.

61. Kohsuke Hayamizu et al, Effects of garcinia cambogia (Hydroxycitric Acid) on visceral fat accumulation ; a double blind, randolized, placebo – controlled trial, Current Therapeutic Research. 2003; 64(8):551-6

ANNEXURES

Proforma

PREVALENCE OF HYPERLIPIDEMIA AMONG HEALTHY FEMALES AND THEIR RELATIONSHIP WITH WAIST CIRCUMFERENCE AND BODY MASS INDEX

SL.NO:

IP.NO:

NAME OF THE PATIENTS ATTENDER:

DOE:

AGE:

SEX:

OCCUPATION:

ADDRESS:

PRESENTING COMPLAINTS IF ANY:

HISTORY OF PRESENTING COMPLAINTS IF ANY:

PAST HISTORY:

H/O DM YES/NO

H/O HIN YES/NO

H/O IHD YES/NO

H/O Medication if any YES/NO

FAMILY HISTORY:

Type-II DM,HTN,IHD

PERSONAL HISTORY:

Diet – veg / mixed

Appetite – good/poor

Sleep – sound/disturbed

Marital status – married/unmarried

Bowel and Bladder – regular/ altered

Habits

Tobacco chewing **:** yes / no Duration

Alcoholic: yes / no Duration Amount

OBSTETRIC & MENSTRUAL HISTORY:

No of Children:

LMP:

Cycles- regular/irregular.

PHYSICAL EXAMINATION:

1. State of nutrition: over weight / normal / underweight

   Wt kg

   Ht mtrs

   BMI kg/m^2

2. Waist circumference in cm:
3. Hip circumference in cm:
4. Waist to Hip ratio:
5. Hydration – well hydrated / dehydrated
6. Eyes-
7. Pupils: Reactive/Non reactive
8. Oral cavity

9. Waist circumference in cm:
10. Hip circumference in cm:
11. Waist to Hip ratio:
12. Hydration – well hydrated / dehydrated
13. Eyes-
14. Pupils: Reactive/Non reactive
15. Oral cavity
16. Clubbing
17. Cyanosis
18. Lymphadenopathy
19. Pedal edema
20. Skin

VITALS:

Pulse

Rate

Rhythum – regular / irregular

Volume – normal/ high / low

Character

Condition of the vessel wall

Peripheral pulse felt / not felt R-R/R-F delay

Blood pressure:

Supine

Standing

Temperature F

Respiratory rate

SYSTEMIC EXAMINATION:

CVS:

Inspection

Palpation

Percussion

Auscultation

ABDOMEN:

Inspection

Palpation

Percussion

Auscultation

RS:

Inspection

Palpation

Percussion

Auscultation

CNS:

Mental status examination
Cranial Nerves
Motor System
Cerebellar examination
Meningeal signs

CLINICAL DIAGNOSIS:

INVESTIGATIONS:

Lipid profile:

Total cholesterol :

Triglycerides :

LDL :

HDL :

VLDL :

Blood sugar: FBS

Urine routine:

Alb :

Sugar :

Microscopy :

ECG in all leads: Rate Rhythm Axis P-Wave P-R Interval QRS Complex ST segment T-Wave U wave

CONCLUSION

Signature of Guide Signature of Student

CONSENT FORM

I am **Dr.Nirmala.P**, carrying out a study on the topic, "**PREVALENCE OF HYPERLIPIDEMIA AMONG HEALTHY FEMALES AND THEIR RELATIONSHIP WITH WAIST CIRCUMFERENCE AND BODY MASS INDEX"**

My research project guide is **Dr.S.Usha M.D.,** My research project is being carried out under the department of General Medicine, Coimbatore Medical College Hospital.

RESEARCH BEING DONE:

"PREVALENCE OF HYPERLIPIDEMIA AMONG HEALTHY FEMALES AND THEIR RELATIONSHIP WITH WAIST CIRCUMFERENCE AND BODY MASS INDEX"

PURPOSE OF RESEARCH

1. To analyse the accuracy of retinoscopy in acceptance of subjective refraction.
2. To analyse accuracy of Autorefraction in acceptance of subjective refraction.
3. To compare the accuracy of retinoscopy and autorefraction in acceptance of sobjective refraction.

SAMPLE SIZE:

100 Healthy Females

STUDY PARTICIPANTS:

Aged 25-60 years asymptomatic healthy females

LOCATION:

Master Health Check up, CMCH, Coimbatore.

PROCEDURES INVOLVED:

The research includes Anthropometric indices – Calculation of Body Mass Index, Waist Circumforence, Waist Hip Ratio, Blood Pressure Measurement, Serum Lipid Profile, Fasting Blood Sugar, Electro Cardiogram and detailed history taking including, Past, Personal, Family & Diet History.

You, Shri./ Smt./ Kum. _____, aged ____ years, D/o / W/o _____, residing at _____ _____ are requested to be a participant in the research study titled "**PREVALENCE OF HYPERLIPIDEMIA AMONG HEALTHY FEMALES AND THEIR RELATIONSHIP WITH WAIST CIRCUMFERENCE AND BODY MASS INDEX"** in Government Medical College Hospital, Coimbatore. You satisfy eligibility criteria as per the inclusion criteria. You can ask any question or seek any clarifications on the study that you may have before agreeing to participate.

DECLINE FROM PARTICIPATION

You are hereby made aware that participation in this study is purely voluntary and honorary and that you have the option and the right to decline from participation in the study.

PRIVACY AND CONFIDENTIALITY

You are hereby assured about your privacy. Privacy of subject will be respected and any information about you or provided by you during the study will be kept strictly confidential.

AUTHORIZATION TO PUBLISH RESULTS

Results of the study may be published for scientific purposes and/or presented to scientific groups, however you will not be identified; neither will your privacy be breached.

STATEMENT OF CONSENT

I, _____, do hereby volunteer and consent to participate in this study being conducted by Dr.Nirmala.P, I have read and understood the consent form / or it has been read and explained to me in my own language. The study has been fully explained to me, and I may ask questions at any time.

Signature / Left Thumb Impression of the Volunteer Date:

Signature and Name of witness Date:

ஒப்புதல் படிவம்

பெயர் :

பாலினம் : வயது:

முகவரி :

அரசு கோவை மருத்துவக் கல்லூரியில் பொது மருத்துவத்துறையில் பட்ட மேற்படிப்பு பயிலும் மாணவி மரு.பெ.நிர்மலா அவர்கள் மேற்கொள்ளும் **"ஆரோக்கியமான பெண்கள் மத்தியில் பரவியுள்ள அதிகபடியான கொழுப்புச்சத்து, இடுப்பு சுற்றளவு மற்றும் உடல் நிறை குறியீட்டெண்"** சம்பந்தமான ஆய்வில் செய்முறை மற்றும் அனைத்து விளக்கங்களையும் கேட்டுக் கொண்டு எனது சந்தேகங்களை தெளிவுபடுத்திக் கொண்டேன் என்பதை தெரிவித்துக் கொள்கிறேன். நான் இந்த ஆய்வில் முழு சம்மதத்துடன் மற்றும் சுய சிந்தனையுடன் கலந்து கொள்ள சம்மதிக்கிறேன்.

இந்த ஆய்வில் என்னுடைய அனைத்து விவரங்கள் பாதுகாக்கப்படுவதுடன் இதன் முடிவுகள் ஆய்விதழில் வெளியிடப்படுவதில் ஆட்சேபனை இல்லை என்பதை தெரிவித்துக் கொள்கிறேன். எந்த நேரத்திலும் இந்த ஆய்விலிருந்து நான் விலகிக் கொள்ள எனக்கு உரிமை உண்டு என்பதையும் அறிவேன்.

கையொப்பம்/ரேகை

இடம் :

நாள் :

MASTER CHART

SL NO	NAME	OP.NO	AGE	OCCUPTN	PH	FH	PRH	DIET	T	A	WT	HT	BMI	WC	TC	HDL	TG	LDL	FBS	BP	WHR
1	Malar	15377	27	HW	N	DM	NS	M	N	N	56	158	22.48	78	165	45	130	94	98	110/80	0.84
2	Dhanam	14856	29	HW	N	NS	NS	M	N	N	56	153	23.92	77	154	44	128	84	97	120/70	0.82
3	Roslin	15677	26	HW	N	NS	NS	M	N	N	45	150	20	75	165	48	128	91	87	110/76	0.76
4	Nagammal	16778	25	HW	N	NS	NS	M	N	N	68	167	24.46	76	152	45	108	85	88	110/70	0.88
5	Reshmi	17886	26	BNS	N	NS	NS	M	N	N	45	148	20.54	79	169	47	125	97	90	120/80	0.86
6	Rithika	15887	27	TAILOR	N	NS	NS	V	Y	N	66	162	25.19	82	212	34	162	145	89	110/82	0.9
7	Savitha	15634	29	GS	N	NS	NS	M	Y	N	58	158	23.29	76	152	32	115	97	97	130/70	0.76
8	Lakshmi	15785	28	COOLI	N	NS	NS	M	N	N	52	156	21.39	77	145	40	134	78	98	126/80	0.79
9	Rekha	17888	27	HW	N	NS	NS	M	N	N	62	158	24.89	75	148	45	125	78	94	130/70	0.91
10	Radhika	16509	28	AG	N	HTN,DM	NS	M	N	N	58	156	23.8	73	158	46	121	88	89	120/70	0.88
11	Latha	18978	29	GS	N	DM	NS	V	Y	Y	68	162	25.95	86	221	36	158	153	139	110/78	0.87
12	Bindhu	16561	30	COOLI	N	NS	NS	M	Y	N	54	156	22.22	75	158	38	116	97	97	120/70	0.77
13	Priya	15643	29	HW	N	NS	NS	M	Y	N	55	160	21.48	76	174	51	125	98	82	110/84	0.76
14	Indhu	14352	26	BNS	N	NS	NS	M	N	N	54	156	23.8	77	169	47	121	98	85	110/80	0.78
15	Geetha	15634	27	TEACHER	N	NS	NS	M	N	N	54	156	22.22	76	158	46	128	86	88	120/80	0.75
16	Shobha	18976	29	LABOUR	N	NS	NS	M	Y	N	55	160	21.48	78.5	151	31	128	94	86	110/80	0.76
17	Banu	17654	29	HW	N	NS	NS	M	N	N	58	168	20.56	77.5	164	45	118	95	88	120/76	0.75
18	Parvathy	14009	26	HW	N	NS	NS	M	N	N	56	160	21.87	78	154	42	128	86	91	110/84	0.86
19	Manjula	15897	28	HW	N	NS	NS	M	N	N	62	158	20.56	79	159	46	118	89	86	110/80	0.84
20	Prema	17658	40	GS	N	HTN	NS	V	Y	N	57	147	26.37	83	230	32	164	165	85	110/70	0.9
21	Swapna	16543	37	BNS	N	NS	NS	M	N	N	51	151	22.36	75.5	174	54	121	96	82	120/80	0.88
22	Usha	14532	36	TEACHER	N	NS	NS	M	N	N	56	160	21.87	76.5	152	44	126	83	89	130/70	0.9
23	Manjula	14543	29	AG	N	NS	NS	M	N	N	62	158	24.83	77	161	45	120	92	87	120/70	0.75
24	Kannammal	14657	35	HW	N	DM,IHD	NS	V	Y	N	72	160	28.12	83	226	34	158	160	118	136/90	0.91

25	Saradha	14689	29	GS	N	DM	NS	M	N	N	55	147	25.45	76.5	151	32	112	97	94	120/80	0.9
26	Sudha	14588	37	HW	N	NS	NS	M	N	N	54	159	22.22	77	156	45	110	89	93	110/70	0.78
27	Kasthuri	15432	26	TEACHER	N	NS	NS	M	N	N	56	160	21.87	78.5	170	48	125	97	91	110/80	0.76
28	Shanthi	16543	39	SA	N	HTN	NS	M	Y	Y	66	150	26.66	82	232	35	166	164	89	120/80	0.86
29	Anitha	13692	27	HW	N	NS	NS	M	N	N	42	154	17.7	77.5	158	32	135	99	90	130/80	0.92
30	Ganga	18765	35	LABOUR	N	NS	NS	M	N	N	56	158	22.48	74.5	161	42	118	95	95	130/80	0.92
31	Malathi	16587	29	HW	N	NS	NS	M	N	N	60	162	22.9	75	158	42	128	90	94	110/70	0.91
32	Mariyammal	18764	36	GS	N	IHD	NS	V	Y	Y	69	157	28.04	84	244	36	164	175	132	140/90	0.92
33	Bashirabanu	17609	27	SA	N	NS	NS	M	N	N	51	151	22.36	76.5	165	51	126	89	108	120/80	0.86
34	Savihri	16709	40	TAILOR	N	NS	NS	M	N	N	60	158	24.09	78	155	43	116	89	106	130/80	0.84
35	Rathna	14500	29	COOLI	N	NS	NS	M	N	N	48	153	20.5	78.5	161	42	118	95	96	120/80	0.86
36	Mumthaj	13456	29	HW	N	DM	NS	M	Y	N	68	160	26.56	79	154	34	166	95	98	110/70	0.91
37	Vanitha	19087	25	NURSE	N	NS	NS	M	N	N	58	162	22.13	78.5	162	41	122	97	102	120/80	0.88
38	Reetu	18769	37	TEACHER	N	HTN	NS	V	Y	N	74	168	26.24	83	228	32	168	162	96	110/70	0.9
39	Jayaprabha	16755	40	GS	N	NS	NS	V	N	N	60	158	24.09	84	220	33	170	183	108	110/80	0.88
40	Devaki	17780	27	SA	N	NS	NS	M	N	N	41	155	17.08	78.5	163	44	122	95	95	110/80	0.88
41	Govindamma	14563	32	SA	N	NS	NS	M	N	N	55	157	22.35	75.5	161	42	122	95	91	120/84	0.76
42	Chandra	15643	25	CHEF	N	NS	NS	M	N	N	52	158	20.88	76	155	40	123	90	90	110/80	0.84
43	Kala	16785	34	LABOUR	N	NS	NS	M	N	N	56	158	22.48	77.5	163	45	135	91	99	110/70	0.86
44	Yamuna	19656	26	HW	N	NS	NS	M	N	N	62	158	24.8	78	169	45	133	97	98	130/70	0.9
45	Hema	16789	37	TAILOR	N	HTN	NS	M	Y	Y	60	144	28.98	84	224	32	172	157	142	140/88	0.88
46	Ananthi	14987	26	HW	N	NS	NS	M	N	N	63	160	24.6	78.5	160	42	113	95	106	110/80	0.88
47	Sahira	15623	28	MANAGER	N	NS	NS	M	N	N	63	160	24.6	79	157	46	125	86	102	120/80	0.87
48	Sundaram	17654	35	GS	N	DM	NS	M	Y	N	58	148	26.48	83	232	35	174	162	109	110/80	0.91
49	Pushpam	15467	37	GS	N	NS	NS	M	N	N	62	162	23.6	77.5	163	42	129	95	98	110/70	0.86
50	Lalitha	16754	26	HW	N	NS	NS	M	N	N	42	150	18.67	77.5	158	35	119	99	89	120/84	0.91
51	Parimala	15689	39	TEACHER	N	HTN	NS	M	N	N	62	152	26.84	76.5	168	47	120	97	123	110/80	0.89
52	Indirani	18976	39	CHEF	N	NS	NS	M	Y	N	62	158	24.8	84	165	45	119	96	96	120/84	0.77
53	Ashabi	18792	27	HW	N	NS	NS	M	N	N	56	163	21.07	77.5	155	35	123	95	105	110/70	0.84

54	Mani	18923	35	GS	N	NS	NS	M	N	N	56	160	21.8	77	164	46	129	92	102	120/80	0.9
55	Shanthi	18543	34	SA	N	HTN	NS	V	N	N	78	170	26.98	83	155	34	123	96	99	110/80	0.88
56	Revathi	14588	26	COOLI	N	NS	NS	M	N	N	60	170	20.76	76.5	154	31	120	99	97	120/70	0.86
57	Savithri	20034	26	WAITER	N	NS	NS	M	N	N	62	160	24.21	77	165	45	131	94	99	120/80	0.9
58	Kamala	20056	27	GS	N	DM	NS	M	Y	N	78	160	30.46	78	156	36	156	94	119	130/84	0.94
59	Kanimozhi	20076	26	MANAGER	N	NS	NS	M	Y	N	60	152	25.97	78	154	32	170	97	105	110/80	0.88
60	Raji	22045	27	TAILOR	N	DM	NS	M	N	N	66	150	29.33	78.5	155	31	164	99	109	140/90	0.89
61	Sujatha	22287	34	BNS	N	NS	NS	M	Y	N	56	158	22.48	77.5	159	42	125	92	101	110/84	0.88
62	Chandra	21044	27	CHEF	N	DM	NS	M	N	N	74	168	26.24	82	230	35	158	163	99	110/80	0.91
63	Saraswathi	21055	32	LABOUR	N	NS	NS	M	N	N	58	162	22.13	76.5	164	44	118	96	97	110/70	0.86
64	Bharathi	24034	25	HW	N	NS	NS	M	N	N	56	157	22.76	77.5	158	41	130	91	98	110/70	0.87
65	Haseena	25013	33	HW	N	NS	NS	M	N	N	60	160	23.43	78.5	155	43	116	89	90	120/80	0.88
66	Reeta	24567	28	DHOBI	N	NS	NS	M	N	N	53	158	21.28	78	158	34	126	99	89	128/70	0.84
67	Kiruthika	22018	35	BNS	N	DM	NS	M	Y	Y	56	154	23.61	82	154	30	125	99	88	120/80	0.9
68	Dhanalakshmi	22756	27	GS	N	NS	NS	M	N	N	60	156	24.65	77.5	164	47	121	93	88	110/80	0.88
69	Anushiya	26544	28	GS	N	NS	NS	M	N	N	56	155	23.3	77	163	42	128	95	94	130/80	0.9
70	Bakkiyam	27698	29	TAILOR	N	IHD	NS	M	N	N	68	160	26.56	83	220	37	162	151	118	120/80	0.91
71	Gowthami	27756	27	TEACHER	N	NS	NS	M	N	N	58	156	23.83	78.5	160	36	126	99	96	110/70	0.88
72	Lakshmi	23389	28	WAITER	N	DM	NS	M	N	N	52	156	21.36	81	213	38	156	144	98	110/80	0.86
73	Narmatha	26656	29	DHOBI	N	NS	NS	M	N	N	45	150	20	77.5	158	35	120	99	105	120/80	0.88
74	Uma	28765	40	SA	N	NS	NS	M	Y	N	70	168	24.82	79	155	32	121	99	103	110/80	0.92
75	Vishalatchi	25400	37	GS	N	NS	NS	M	N	N	56	158	22.48	79.5	155	42	124	88	105	120/80	0.91
76	Sunitha	21987	38	TEACHER	N	NS	NS	M	N	N	60	162	22.9	79	166	45	125	96	96	110/80	0.92
77	Sanmathi	18765	28	CHEF	N	HTN	NS	M	N	N	56	158	22.48	84	217	36	164	148	133	120/80	0.88
78	Aswini	15473	27	HW	N	NS	NS	M	N	N	52	158	20.88	79.5	155	32	118	99	105	110/80	0.9
79	Ranjitham	17864	38	MANAGER	N	NS	NS	M	Y	Y	58	160	22.65	82	162	42	118	96	99	120/80	0.91
80	Muniyal	16543	39	BNS	N	HTN	NS	V	Y	N	68	155	28.33	78	158	42	175	92	118	150/90	0.92
81	Neeraja	18700	29	GS	N	NS	NS	M	N	N	62	172	21.01	83	151	31	125	95	90	110/80	0.76
82	Menaka	18976	35	GS	N	IHD	NS	M	Y	N	68	160	26.56	75.5	161	45	115	93	89	120/70	0.75

83	Manjula	18754	28	HW	N	NS	NS	V	N	N	62	172	21.01	79	153	34	166	95	93	110/70	0.87
84	Haripriya	17653	34	AG	N	NS	NS	V	N	N	68	168	24.11	78.5	165	45	126	95	96	110/80	0.9
85	Padmini	18799	36	SA	N	NS	NS	M	Y	Y	70	160	27.34	83	153	35	170	93	95	120/80	0.92
86	Pangajam	17654	37	TAILOR	N	NS	NS	V	N	N	58	170	20.06	79	153	42	118	87	93	110/80	0.9
87	Gowri	18734	29	TEACHER	N	DM	NS	M	N	N	72	160	28.12	83	208	30	170	144	119	120/80	0.86
88	Ponni	16543	40	GS	N	NS	NS	M	N	N	52	158	20.88	77.5	168	45	120	99	101	110/70	0.88
89	Yasodha	18945	28	SA	N	NS	NS	M	N	N	66	170	22.83	78.5	160	46	138	86	105	110/70	0.89
90	Leelavathy	21765	27	DHOBI	N	NS	NS	M	N	N	72	170	24.9	79	155	34	123	96	99	110/80	0.86
91	Nalini	27058	35	GS	N	HTN	NS	V	Y	Y	66	150	29.33	83	155	34	178	95	106	150/100	0.91
92	Radha	23478	26	HW	N	NS	NS	M	N	N	56	154	23.61	76.5	165	46	122	95	101	120/80	0.84
93	Lakshmi	24536	34	NURSE	N	HTN	NS	V	N	N	69	157	28.04	79.5	159	34	129	99	89	120/80	0.9
94	Malathi	23478	25	TEACHER	N	NS	NS	M	N	N	56	155	23.3	78	158	34	129	98	90	120/70	0.88
95	Renuka	23111	34	CHEF	N	DM	NS	V	Y	N	57	147	26.38	84	160	36	186	98	135	110/80	0.9
96	Suseela	24345	25	GS	N	NS	NS	M	N	N	60	158	24.03	78.5	169	45	129	98	89	120/80	0.87
97	Haseena	24765	26	HW	N	NS	NS	V	N	N	52	154	21.92	79	166	42	130	98	96	120/70	0.89
98	Seetha	18976	32	HW	N	NS	NS	M	N	N	56	157	22.76	79.5	153	45	129	82	98	110/80	0.88
99	Banu	29087	27	HW	N	HTN	NS	M	N	N	78	162	29.77	82	210	30	166	147	99	120/80	0.9
100	Dhanalakshmi	29078	34	GS	N	NS	NS	M	N	N	60	160	23.43	78.5	166	42	131	98	101	110/80	0.86

KEY TO MASTER CHART

1. SL.NO — Serial Number
2. Name
3. IP .No — Inpatient Number
4. Age
5. Sex — M-Male
   F-Female
6. Ocpn — Occupation,
   - STU-Student
   - HW-House Wife
   - GS-Government Service
   - BNS-Business
   - AG-Agriculture
   - SA-Sales Assistant
   - ENG-Engineer
   - Nurse
   - Tailor
   - Dhobi
   - Waiter
   - Teacher
   - Cooli
   - Chef
   - Labour
   - Manager
   - Typist
   - Aya
   - Attender

7. PH	**Past History,**	DM-Diabetes Mellitus,	Y: Yes
			N: No
		HTN- Hypotension,	Y: Yes
			N: No
		IHD- Ischemic heart Disease	Y: Yes
			N: No
		MED-Medication If Any	Y: Yes
			N: No
8. FH	**Family History,**	NS-Nothing significant	
		HTN- Hypotension	
		DM-Diabetes Mellitus	
		IHD- Ischemic heart Disease	
9. PRH	**Personal history**,	NS-Nothing significant	
	Diet	V- Vegetarian	
		M- Mixed	
10. S	Smoking,	Y- Yes	
		N- No	
11. A	Alcoholic	Y- Yes	
		N- No	
12. T	Tobacco chewing	Y- Yes	
		N- No	

General Physical Examination

13. Wt	Weight	
14. Ht	Height	
15. BMI	Body Mass Index	
16. W.C	Waist Circumference	
17. H.C	Hip Circumference	
18. W/H	Waist Circumference	
19. BP	Blood Pressure	Systolic/ Diastolic

Investigations

20. TC	Total Cholesterol
21. TGL	Triglycerides
22. LDL	Low Density Lipoprotein
23. HDL	High Density Lipoprotein
24. FBS	Fasting Blood Sugar